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Proceedings of the British Cardiac Society

At the 66TH ANNUAL GENERAL MEETING of the British Cardiac Society held in Dundee at Ninewells Hospital, on Wednesday and Thursday, 8 and 9 April 1987, E Sowton succeeded D G Julian as President and the following members were elected to Council: A Henderson, I Hutton, A S Hunter, on the retirement of C P Aber, M J Godman, and M Stephens.

The President asked the Society to rise in memory of the following members who died during the previous year: K Shirley-Smith, D Pickering, H Neufeld, A W W B Woods, and A G W Whitfield.

New members joining the Society were as follows: T Aherne (Cork); G Angelini (Cardiff); D B Barnett (Leicester); P Bloomfield (Edinburgh); W Brawn (Australia); J L Caplin (Southampton); J F Cleland (London); A B Davies (Penarth); B M Fabri (Liverpool); R K Firmin (Leicester); J L Gibbs (Bramham); H H Gray (Kingston upon Thames); H M Husaini (Stockport); B S Jenkins (London); A Lahiri (Harrow); B Lewis (London); D P Lipkin (Harrow); H G Love (Altrincham); D P Nicholls (N Ireland); A D C Norris (Faversham); R J Northcote (Glasgow); C R Nyman (Boston); T H Pringle (Belfast); A C Rankin (Glasgow); D B Rowlands (Wirral); A Rozkovec (Bristol); J H Silas (Wirral); P L C Smith (London); N C Taylor (Dyfed); S R Underwood (London); J M Walker (Oxford).

An announcement was made of the importance of the social programme for the Society's Jubilee meeting to be held at Wembley on the 24 to 26 November 1987. There will be plenary sessions on Great British Cardiologists of the Past and Cardiology for the Future.

During the meeting the scientific sessions were held under the chairmanship of H Watson and D Emslie-Smith and the following are abstracts of the papers that were presented.

Balloon aortic valvuloplasty: high frequency of early recurrence of aortic stenosis

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Eight patients aged 68 to 89 underwent nine balloon aortic valvuloplasty procedures, three from the brachial and the remainder from the femoral route. Balloon sizes ranged from 1·2 cm to 2·1 cm triple lumen and two patients had the double balloon technique. A significant (>30%) fall in peak to peak aortic valve gradient was recorded immediately after eight procedures. The calculated aortic valve area (thermodilution cardiac output) increased in five out of six patients. Doppler estimates of aortic valve gradients were made before and one to two days after the procedure and showed significant improvement (>30%) in only one patient. This patient was the only one to show sustained symptomatic benefit. Another had relief of angina for two days. One patient had a small stroke two hours after the procedure. Five patients subsequently had aortic valve replacement. Three valves were bicuspid and two were tricuspid. No evidence of valve disruption was apparent at operation. In two patients the epicardial surface of the left ventricle was bruised: in one bruising on the anterior surface was associated with a large pericardial haematoma and in the other the bruise was at the apex. In the patient who had suffered the stroke there was thrombus at the apex of the left ventricle. Operation was complicated by anterior myocardial infarction in one patient and a major stroke in another.

We conclude that balloon aortic valvuloplasty is a relatively safe procedure but that it may produce only temporary deformation of the aortic valve orifice which quickly returns to its original shape. Improvement in haemodynamic function during the procedure does not imply lasting benefit.

Valvuloplasty for mitral stenosis in middle aged and elderly patients

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Percutaneous transvenous balloon dilatation of mitral valve stenosis was attempted in eight patients

aged 38–74 (mean 67). In one patient with giant left atrium (dimension 14 cm) the balloon catheter could not be made to cross the septum: in seven patients balloon dilatation was carried out with a single 25 mm balloon in two, a 25 mm trefoil balloon in four, and double balloons in one. The major technical difficulty was that of achieving the optimal position of the balloon at the mitral valve orifice. Cross sectional echocardiography proved useful for positioning 3 cm length balloons, particularly in patients with a dilated left ventricle. In four patients it was necessary to advance the guide wire to the descending aorta to provide stability for the balloon to turn within the enlarged left atrium. In two symptomatic patients with moderate mitral stenosis there was only a very small reduction in resting gradient. In four patients with more severe stenosis the immediate reduction in gradient was from 12·4 to 6·4 mm Hg. Preliminary results suggest that the improvement in gradient and valve area was maintained at repeat catheterisation 2–4 months later. When positioned at the mitral orifice, balloon dilatation caused a considerable drop in aortic pressure and a slight rise in pulmonary artery pressure, but only one patient developed brief syncope. The trefoil shaped balloon did not prevent a considerable drop in systolic pressure and had the disadvantage of tending to kink, sometimes thereby preventing full deflation. Mitral valvuloplasty is an effective alternative to surgical valvotomy even in older patients.

Residual stenosis after percutaneous transluminal coronary angioplasty and its effects on left ventricular function: evidence for delayed healing at angioplasty site

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Percutaneous transluminal coronary angioplasty (PTCA) provides effective symptomatic relief in selected patients with angina. Nevertheless, the effects of the procedure on left ventricular function are not well defined. We prospectively studied 86 patients with single vessel coronary artery disease treated by PTCA. Thallium-201 perfusion scintigraphy and technetium-99^{m} left ventriculography were used to evaluate myocardial ischaemia and left ventricular ejection fraction (EF), respectively. Imaging was performed before and 3·6 (2·2) months after PTCA. Before PTCA, reversible myocardial ischaemia was present in every case and stress produced dilatation of the left ventricule with a fall in EF from a mean of 64 (6) % to 56 (7) % ($p < 0·001$). After PTCA residual coronary stenosis was $\leqslant 20\%$ in 78 patients (group 1) and 20 to 50% in eight patients (group 2). Symptomatic relief and correction of reversible myocardial ischaemia were similar in both groups but group 2 showed persistent stress-induced left ventricular dilatation with a fall in EF from 65 (6) % to 56 (5) %. In group 1 the left ventricular EF response to stress tended to normalise (63% to 67%, $p < 0·001$) although 30% of the group studied very early (< 6 weeks) after PTCA showed residual decompensation (61 (9) % to 52 (9) %). In this subgroup repeat studies three months later, however, showed complete correction of left ventricular function: resting EF 66%, 69% with stress, and no increase in end diastolic volume.

These date indicate that: (*a*) a residual stenosis of $< 50\%$ after PTCA is associated with a good symptomatic response but detectable left ventricular dysfunction often persists unless the residual stenosis is $\leqslant 20\%$; (*b*) the healing process at the PTCA site may delay functional improvement for up to three months.

Inferior ST segment depression during left anterior descending coronary angioplasty: inferior ischaemia or "reciprocal" change?

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During left anterior descending (LAD) coronary angioplasty inferior ST segment depression frequently occurs in association with anterior ST elevation. It is unclear whether such changes are "reciprocal" or indicate additional inferior ischaemia. Therefore, intravenous digital subtraction left ventriculography was performed before and during balloon inflation to assess ischaemia in 16 patients with single vessel coronary disease undergoing elective LAD angioplasty. A twelve lead electrocardiogram was recorded throughout. During balloon inflation eight patients (group 1) developed inferior ST segment depression (mean 2 mm, range 1–3 mm) associated with anterior ST segment elevation (mean 5 mm, range 2–9 mm). The remaining eight patients (group 2) developed either anterior ST segment elevation alone (three patients) or had no ST alteration in any lead. Left ventricular (LV)

ejection fraction fell during balloon inflation in both groups (group 1, 76% to 48%; $p < 0.001$ and group 2, 73% to 57%; $p < 0.001$), this reduction being greater in group 1 ($p < 0.01$). Regional shortening of the anteroapical LV segment also fell in both groups (group 1, 42% to 12%; $p < 0.001$ and group 2, 42% to 19%; $p < 0.001$). In contrast, regional shortening of the inferobasal LV segment did not change in either group (group 1, 30% to 37%; NS and group 2, 28% to 38%; NS), nor was there a difference between groups.

Inferior ST segment depression during LAD angioplasty, although associated with a more pronounced reduction in global LV performance, was not accompanied by a deterioration in inferobasal LV contraction. Thus such electrocardiographic change represents a reciprocal phenomenon rather than the presence of additional inferior ischaemia.

Pulsed Doppler ultrasound in the diagnosis of post-infarction ventricular septal defect

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Eleven patients with loud systolic murmurs that developed during the course of acute myocardial infarction were referred to the Regional Cardiac Centre. Ten had ventricular septal defects (VSDs) and one had torrential mitral regurgitation. The site of the infarct was anterior in seven and inferior in four. In all but one it was the first infarction. Mean time to transfer was 6·7 days and many patients were in a poor condition on arrival. Cross sectional echocardiography alone demonstrated the VSD in all three patients with inferior infarctions, but in only three of the seven with anterior infarcts, because the septum bulged into the right ventricle (RV). Pulsed Doppler ultrasound, however, demonstrated high velocity turbulence in the RV in all ten patients, and accurately identified the site of the defect in eight. Continuous wave Doppler identified severe mitral regurgitation in the remaining patient. Four patients were sent for operation on the basis of cross sectional and Doppler imaging alone but one died before operation. Seven patients were catheterised; single vessel disease was found in five of them, and only one graft was placed at operation. One patient in whom the operation was initially successful became breathless three months later and a loud murmur was heard. Doppler ultrasound confirmed patch dehiscence, despite essentially unchanged echocardiographic images, and correctly estimated RV pressure.

If good echocardiographic images and Doppler signals can be obtained, cardiac catheterisation is of doubtful additional benefit in these patients.

Analysis of early markers of myocardial infarction

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The use of intravenous thrombolysis requires early and accurate diagnosis of myocardial infarction (MI). This is usually based on clinical assessment and ECG criteria. Serum myoglobin rises in the early phase of MI and may therefore offer additional information. We evaluated 68 patients with significant chest pain 1 hour (mean 6), who presented to the coronary care unit (CCU) within 24 hours of onset of symptoms. A 12 lead ECG was obtained and serum myoglobin, creatine kinase (CK), and CK-MB were measured within an hour. MI was confirmed in 65% of patients by use of the WHO criteria during the first 48 hours. The admission ECG was classified as ST elevation (group I), any ECG abnormality (II), and normal (III). Sixty one per cent of the MI patients were in group I, 32% in group II, and 7% in group III. Corresponding values in the non-MI group were 13%, 71%, and 17% respectively. Mean (SD) total CK and CK-MB activities (144(187) IU/l and 17(24) IU/l respectively) in the MI group were significantly higher than those in the non-MI group (49(37) ($p < 0.02$) and 6(4) ($p < 0.05$) respectively). Serum myoglobin was also higher in the MI group (188(139) μg/l) than in the non-MI group (46(49) μg/l ($p < 0.001$)). There was a good correlation between a radioimmunoassay and a rapid latex agglutination test ($p < 0.001$) that gave a result within 10 minutes for serum myoglobin. By stepwise analysis of the ECG and myoglobin, using group I alone, and group II and III ECG with an abnormal myoglobin (> 90 μg/l) we could identify 82% of patients who would develop a MI on follow up with 17% false positives.

These data suggest that analysis of ECG and serum myoglobin (measured by a rapid latex kit) as early markers of MI could identify the majority of patients who develop MI within an hour of admission to a CCU. This simple and fast stepwise analysis

would be useful in identifying patients for intravenous thrombolytic treatment.

β blockers and the hypokalaemia associated with acute chest pain

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Plasma potassium was measured on admission in 1234 patients who presented with acute ischaemic chest pain. 195 of the patients were on β blockers before admission. Comparison of the potassium concentrations of patients admitted early (within 4 hours of onset of symptoms) with those admitted later (4 to 18 hours) showed that in patients not pretreated with β blockers there was a transient fall in plasma potassium (difference in group mean 0·13 mmol/l, $p < 0·001$). This produced an early transient increase in the occurrence of hypokalaemia (difference in group mean 11%, $p < 0·01$). Neither the fall in potassium nor the increase in hypokalaemia was seen in patients who had been on β blockers before admission. Further, when only patients admitted within 4 hours of onset of symptoms were considered, pretreatment with β blockers was shown to be associated with a higher mean plasma potassium (difference in group mean 0·14 mmol/l, $p < 0·01$) and a lower frequency of hypokalaemia (difference in group mean 16%, $p < 0·01$) than those on no drug treatment. Non-selective β blockers were found to be more effective than cardioselective agents in maintaining plasma potassium concentrations.

These findings suggest a mechanism for the beneficial effects of β blockers on morbidity and mortality in acute myocardial infarction.

Exertional ventricular tachycardia and myocardial ischaemia after coronary artery surgery

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Coronary artery bypass grafting (CABG) improves angina but its effect on exercise induced ventricular arrhythmias is unclear. We examined this issue in 54 male patients who underwent symptom limited treadmill exercise testing one day before and 10 days, one month, and six months after CABG. Exercise haemodynamic function and tolerance increased progressively after CABG. Values at six months were: maximal heart rate 118 to 151 beats/minute ($p < 0·001$); rate-pressure product 16·6 to 27·0 × 1000 ($p < 0·001$); and exercise duration 11·9 to 16·5 minutes ($p < 0·0001$). The frequency of angina (67% to 2% ($p < 0·001$)) and ST segment depression (71% to 20% ($p < 0·001$)) both decreased significantly early after CABG, but in 11 patients ST segment depression developed at six months. The frequency of ventricular arrhythmias recorded at rest, during exercise, and after exercise increased significantly after CABG; 85% of the patients had an exertional ventricular arrhythmia at one month. The occurrence of complex ventricular arrhythmias (couplets or ventricular tachycardia) also increased postoperatively; 18 patients had unsustained exertional ventricular tachycardia at one month. The occurrence of ventricular arrhythmias appeared unrelated to previous myocardial infarction, the extent of coronary artery disease, or the number of grafts inserted.

We conclude that although CABG improves myocardial ischaemia and exercise tolerance, serious ventricular arrhythmia appears to be more common after CABG and unrelated to myocardial ischaemia. This observation may partly explain the reported improvement in symptoms but largely unchanged prognosis in certain groups of patients after CABG.

Long term clinical follow up of patients after coronary surgery and assessment of the benefits of postoperative aspirin and dipyridamole

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A trial of aspirin and dipyridamole (Persantin) after coronary bypass grafting was undertaken between 1978 and 1982. The 320 patients entered have now been further assessed a mean of 6·6 years after operation. Those in the active group took aspirin (300 mg three times a day) and dipyridamole (75 mg three times a day) for a mean 25 months. There were 33 deaths during the follow up and 25 (7·8%) were due to cardiac causes. Fifteen of the 25 cardiac deaths occurred in patients with abnormal preoperative ventriculograms. Thirteen cardiac deaths occurred in the active group and 12 in the placebo group. Five patients refused follow up. Of the 282 patients thus

available for review, 250 attended as outpatients and were assessed with a detailed questionnaire and exercise stress test. One hundred and twenty two had been given active treatment and 128 had been given placebo. Ninety four of the 250 patients complained of angina (49 active, 45 placebo), which had developed a mean 31 months after operation, and 52 of these (28 active, 24 placebo) had abnormal exercise tests. Sixteen patients (6 active, 10 placebo (NS)) required repeat angiography for recurrent symptoms. Eight of the 12 patients (4 active, 8 placebo (NS)) who have had a further operation continue to be symptomatic. Of the nine patients who sustained myocardial infarction during follow up, seven were in the active group. Only nine of the 23 patients with properative hyperlipidaemia and 23 of 66 postoperative smokers were among the 94 patients with recurrence of symptoms. Thus 6·6 years after vein grafting 156 (62·4%) of 250 patients were symptom free and 104 (41·6%) were in full time work.

Administration of aspirin and dipyridamole for a mean 25 months after coronary bypass grafting does not confer long term clinical benefit.

Neuropsychological sequelae of bypass twelve months after coronary artery surgery

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We have reported to the society the short term (8 week) cerebral consequences of coronary artery bypass surgery (CABS) compared with a group undergoing major vascular surgery. We have now completed one year assessments on a cohort of 66 CABS patients previously evaluated before operation and eight days and eight weeks after operation. Ten tests, designed to be sensitive to minor diffuse cortical impairment, were administered on each of these occasions. Neuropsychological deficit was considered to be present when the postoperative performance showed a deterioration by at least one standard deviation (SD) from the patient's preoperative measure in two or more tests. Seventy three per cent (70% confidence interval, 66%–79%) showed a deficit at eight days, improving to 38% (70% confidence interval, 31%–45%). There was no further improvement at 12 months when 35% (70% confidence interval, 28%–42%) still showed impairment. Neuropsychological deficit was strongly associated with age (mean (SD) 59 (6·6) *vs* 53 (7·8) years; Mann-Whitney $U = 697$, $p < 0·01$), longer bypass time (94 (22) *vs* 83 (27) min; $U = 623$, $p < 0·05$), and with operation time (263 (58) *vs* 236 (61) min; $U = 644·5$, $p < 0·05$).

These findings indicate that the cortical effects of CABS are related to the magnitude of surgery and the age of the patient and persist for at least 12 months after operation.

Determinants of patient status ten years after coronary bypass grafting for angina

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Between October 1969 and December 1975, 428 patients underwent isolated coronary artery bypass grafting (CABG) for angina at Harefield Hospital. Thirty six patients were lost to follow up, leaving 392 (92%) for follow up over a minimum period of 10 years (mean 11·5 years). Perioperative mortality was 3·6% and reoperation before 10 years was performed in 15 (3·8%) patients. Five and 10 year survival rates were 85% and 67% respectively with an annual mortality rate of 2·5% for the first five years and 3·0% between five and 10 years. We studied the influence of the patient's age, sex, coronary risk factors, previous infarction, coronary anatomy, left ventricular function, use of internal mammary artery graft, endarterectomy, perioperative infarction, and graft patency at one and five years after operation on the survival at ten years. Late survival was reduced by age >60, hypertension, multivessel disease, and impaired left ventricular function, and improved by full graft patency and possibly by use of an internal mammary artery graft. Of the 273 survivors at 10 years, 100 (38%) were symptom free without a cardiovascular event (that is myocardial infarction or reoperation). One hundred and twenty (46%) had varying degrees of angina, 59 (22%) had clinical evidence of heart failure, and 31 (11·8%) had late myocardial infarction. Of the possible determinants of symptomatic status studied, hypertension increased the incidence of angina, and poor left ventricular function increased the incidence of heart failure. Full graft patency increased the proportion of symptom free survivors.

It is hoped that these data will be useful in predicting and optimising late results of CABG.

Functional assessment of internal mammary artery grafts

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Internal mammary artery grafts to the left anterior descending coronary artery have been shown to have favourable patency rates, but the ability of these grafts to respond to stress is less clear. A group of 15 symptom free patients with patent internal mammary artery grafts at a mean of two years were investigated. Regional myocardial nutrient flow was assessed from the clearance of xenon-133 by a gamma camera fitted with a specially designed high sensitivity biplane collimator, which allowed simultaneous acquisition of 30° and 70° left anterior oblique projections. After direct injection into the graft, left ventricular flow was measured at rest, during atrial pacing at 100 beats/minute, and during atrial pacing at a rate similar to that during maximal exercise (mean 124 beats/minute). In six patients the diameter of the grafted vessel was smaller than the internal mammary artery graft and in these patients mean resting myocardial flow was 45·7 (5·0) ml/100 g/min, falling with atrial pacing at 100 beats/minute to 36·9 (6·4) ml/100 g/min ($p < 0·05$) and falling further to 31·2 (6·2) ml/100 g/min ($p < 0·02$) at the higher pacing rate. In nine patients the diameter of the grafted vessel was similar or larger than that of the graft. Mean resting flow was 52·8 (3·9) ml/100 g/min and was maintained with pacing at 100 beats/minute (53·3 (8·7) ml/100 g/min (NS)), and increased on pacing at 124 beats/minute to 58·5 (5·8) ml/100 g/min ($p < 0·05$).

These results suggest that despite the favourable angiographic appearance the capacity of an internal mammary artery graft to respond to stress is dependent on the interface of the implant to the grafted vessel.

Low energy level internal defibrillation during cardiopulmonary bypass

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High energy electrical shocks damage the myocardium. Nevertheless, it is still common practice to use 20–30 J shocks to defibrillate hearts during cardiopulmonary bypass: some defibrillators cannot be set to deliver less than 10 J. We have modified a standard Albury Lifeguard Defibrillator to deliver energy in small increments starting at 1 J and proceeding to 2, 4, 6, 8, 9, 12, 15, 20, and 30 J. During the period 1983–85, 168 patients (118 M and 50 F; median age 58, range 4–77) required defibrillation on cardiopulmonary bypass. They were successfully defibrillated with the modified defibrillator. Eighty eight (52·3%) patients underwent coronary artery grafting; 59 (35·1%) had valves replaced (22 aortic, 33 mitral, and 4 double valves); and 12 of the remaining 21 patients had correction of congenital defects.

Seventy eight (46%) patients were defibrillated with 2 J or less. In 139 (82·7%) patients defibrillation was effected with 4 J or less. Only four patients required > 10 J to defibrillate their heart.

We recommend the use of low energy defibrillation as a means of preventing electrical damage to the myocardium.

The role of peroperative hypocapnia in the aetiology of neurological deficit after cardiopulmonary bypass surgery

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Thirty patients undergoing coronary artery bypass surgery had a detailed preoperative and postoperative ophthalmological and neuropsychological assessment. Anaesthetic and surgical protocols were standardised, although ventilatory variables were left to the discretion of the anaesthetist and as such closely conformed to accepted clinical practice. A continuous recording of mean arterial pressure, jugular venous pressure, and cerebral perfusion pressure was obtained peroperatively. Blood gas analysis was performed at regular intervals peroperatively. Although none of the patients complained of visual symptoms after operation seven (28%) were found to have ophthalmological signs highly suggestive of ocular hypoperfusion. No intraluminal emboli or evidence of branch vessel embolisation was noted. Examination of the results of the peroperative monitoring showed that the group showing positive postoperative ophthalmological signs had: (*a*) significantly lower arterial carbon dioxide ($p < 0·001$) and oxygen ($p < 0·01$) tensions at the onset of cardiopulmonary bypass (CPB); (*b*) a significantly greater rise in both $PaCO_2$ ($p < 0·01$), and jugular venous pressure ($p < 0·01$) at the onset of CPB; (*c*) a significantly lower mean cerebral perfusion pressure ($p < 0·01$) during the first ten minutes of CPB, a period not maximally covered by the cerebral protecting capabilities of hypothermia. In addition they showed significantly a greater postoperative neu-

ropsychological deficit (80%) than the group who showed no new ophthalmological signs (40%).

Right and left ventricular function after anatomical correction and the Mustard and Senning operations for transposition of the great arteries

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After definitive surgery for transposition of the great arteries (TGA) late morbidity and mortality can be associated with ventricular dysfunction. We examined right and left ventricular function by first pass and equilibrium gated nuclear angiography in 21 patients after anatomical correction of TGA and in 21 patients after intra-atrial repair of TGA by the Mustard (11 patients) and Senning (10 patients) operations. The mean age of the patients after anatomical correction was 51·8 (range 17–85) months and the studies were performed a mean of 47·1 (range 17–78) months after operation. Thirteen patients had undergone primary correction (nine as a neonate and four with ventricular septal defect) and eight had preliminary pulmonary artery banding as part of a two stage repair. The mean age of the patients after intra-atrial repair of TGA was 82·8 (range 13–228) months and studies were performed a mean of 67·4 (range 3–187) months after operation. After injection of technetium-99m labelled autologous erythrocytes, the right ventricular ejection fraction (RVEF) was determined from the first pass study in the anterior projection and the left ventricular ejection fraction (LVEF) from an equilibrium study in the left anterior oblique projection. After anatomical correction the mean RVEF was 57·9% (11·99) and after intra-atrial repair it was 51·1% (10·54). After anatomical correction the mean LVEF was 58·2% (8·68) and after intra-atrial repair it was 57·7% (10·99). The difference in systemic ventricular ejection fraction between the two groups is not statistically significant ($0·05 < p < 0·1$); however, moderately severe ventricular dysfunction ($EF \leqslant 30\%$) was only seen after intra-atrial repair (two patients).

Total anomalous pulmonary venous drainage: a seventeen year surgical experience

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Between 1968 and 1985, 80 children underwent repair of total anomalous pulmonary venous drainage (TAPVD). There were 47 boys and 33 girls (aged from 3 days to 16 years, mean 7·7 months). Seventy (88%) of the children were < a year old at operation. Fifty eight (73%) weighed < 5 kg (range 1·6–42 kg, mean 4·9 kg). Forty five (56%) children had supracardiac, 14 (18%) cardiac, 15 (19%) infracardiac, and six (7%) mixed TAPVD. Pulmonary venous obstruction was present in 27 (34%). The pulmonary artery (PA) peak systolic pressure ranged from 24 to 140 mm Hg (mean 62) and the RV:LV peak systolic pressure ratio from 0·38 to 1·8 (mean 0·88). There were 14 (17·5%) early deaths, of which seven (15%) occurred in the supracardiac, two (14%) in the cardiac, four (26%) in the infracardiac, and one (16%) in the mixed TAPVD groups. During a mean follow up of 65 months (range 6–189 months) there were six (7·5%) late deaths. Multivariate analysis showed no significant influence of sex, age at operation, weight at operation, anatomical type, the presence of pulmonary venous obstruction, or the technique of operation. Postoperative pulmonary venous obstruction occurred in five (6%) patients between six weeks and nine months after operation. All five died, three after reoperation. The surviving patients were all symptom free.

Early repair of TAPVD is feasible irrespective of age and gives excellent long term results. The recurrence of pulmonary venous obstruction after operation, however, is an important adverse factor influencing the late results.

Results of surgery for interrupted aortic arch in infancy: a multivariate and logistic analysis

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Between 1971 and 1986, surgery was performed in 23 infants with interrupted aortic arch (IAA) (11 boys and 12 girls; weight 1·7–4·2 kg (mean (SD); 3·1 (0·63); age 2–90 days (13 (18·7)). Arch interruption was type A in four (17%) patients, type B in

17 (74%), and type C in 2 (9%). Associated cardiac lesions included transposition of the great arteries (2), truncus arteriosus (1), tricuspid atresia (1), double inlet ventricle (1), aortopulmonary window (1), and ventricular septal defect (VSD) (23). The arch was reconstructed with a prosthetic conduit in 12 patients, by direct anastomosis in six, with the left subclavian artery in three, and with the ductus arteriosus in two. Four patients had simultaneous closure of a VSD; pulmonary artery banding was performed in 15 of the remaining 19. Of the 13 (57%) operative deaths, five occurred in patients with complex cardiac defects and three when single stage repair was attempted. Survival after a palliative procedure in patients with VSD was 62% and six of these patients subsequently underwent pulmonary artery debanding and VSD closure with one death. χ_2 and t tests of 13 variables showed that only the year and the type of operation were significantly associated with improved survival. Multiple logistic regression identified male sex, preoperative prostaglandin infusion, absence of cardiac catheterisation, and older age at operation as favourable predictors.

The two stage repair with interposition of a conduit from ascending to descending aorta now offers good palliation for infants with IAA and VSD.

The changing role of duplex scanning in the preoperative evaluation of congenital heart disease

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Previous studies on the usefulness of ultrasound in the preoperative assessment of congenital heart disease have been restricted to small selected patient populations. The potential benefits and problems inherent in surgical referral based solely on clinical or ultrasound studies or both have not been defined in terms of a total paediatric cardiology practice. Furthermore, no study has investigated what additional benefits may have accrued from the introduction of integrated Doppler echocardiography. To this end we reviewed the total population (4289 new referrals) of a regional paediatric cardiology unit over a seven year period (1980–86). 1094 children underwent cardiac surgery (669 bypass, 425 closed), 240 of whom had not had preoperative catheterisation (10% bypass, 41% closed). The overall catheter:operation ratio (C:O ratio) fell progressively from 1:1 in 1980 (both bypass and closed) to 0·42:1 in 1986 (bypass 0·76:1, closed 0·3:1). Further analysis of three main groups (*a*) neonates, (*b*) infants, and (*c*) children aged 1–14 years indicated that the main impact had been on the neonatal and infant groups where cardiac morphology was the main determinant of operation without catheterisation (1986—neonatal C:O ratio, bypass = 0·27:1, closed = 0·0:1; infant C:O ratio, bypass = 0·76:1, closed = 0·42:1). Statistical analysis confirmed that Doppler echocardiography increased the non-invasive referral rates in all three groups and had its greatest effect in defining outflow tract gradients. During the study there were three important ultrasound misdiagnoses (1·3% total) with no resultant surgical mortality, a level of inaccuracy comparable to that in the catheterised group (0·9% total).

With the introduction of Doppler echocardiography non-invasive assessment is increasingly a practical alternative to preoperative catheterisation in well defined subsets of a total paediatric cardiology practice.

Transcatheter occlusion of persistent ductus arteriosus

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Transcatheter occlusion of persistent ductus arteriosus is an attractive alternative to operation. We have successfully implanted occlusion devices in 10 patients aged 1–63 years using Rashkind (USCI) transcatheter occlusion system. The percutaneous approach was used in all—via the femoral vein in nine and the femoral artery in one. From the vein a long sheath was advanced to the aortic opening of the ductus arteriosus whereas from the arterial approach the sheath was advanced to the pulmonary artery opening. The delivery system and occluding device were then advanced, and by manipulation of the delivery system and sheath the device was positioned so that it straddled the pulmonary opening of the ductus arteriosus. It was then released. The PDA was occluded by a 12 mm device (8 French delivery system) in six patients and by a 17 mm device (11 French delivery system) in four patients. In one patient the occluding device was incorrectly positioned and was released into the aorta. It lodged at the aortic bifurcation from where it was retrieved with an ureteric basket catheter and removed via a right femoral arteriotomy. Six weeks later, at the patient's request, successful transcatheter occlusion was performed. Doppler evaluation 24 to 48 hours

after occlusion showed absence of ductal flow in eight and reduced ductal flow in two patients.

Successful transcatheter occlusion of a ductus arteriosus can be achieved in a wide age range of patients. Complications in relation to the accurate placement of the device were encountered in the learning phase.

Balloon angioplasty as first-line treatment for aortic coarctation

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Although balloon angioplasty is widely used for the treatment of postoperative recoarctation of the aorta, its role as first-line treatment for native coarctation has yet to be established. We present our results with balloon dilatation of native coarctation in 13 patients aged 1–19 (mean 11) years. Nine (69%) were hypertensive on presentation. Under general anaesthetic the systolic pressure gradient across the coarctation was 24–50 (mean 30) mm Hg. The aortic diameter above the coarctation was 6–23 (mean 14) mm and at the coarctation site it was 4–9 (mean 6) mm. The size of balloon catheter used was based on the diameter of the distal aortic arch. Meditech balloon catheters, 8–18 mm in diameter, were inflated 1–4 (mean 2·1) times at 60–110 psi. The ratio of balloon:coarctation diameter in individual patients was 2·0–4·5 (mean 2·7). After angioplasty the systolic pressure gradient fell to 0–20 (mean 6) mm Hg ($p<0·01$) and the coarctation diameter increased to 7–20 (mean 12) mm ($p<0·01$). Only one patient developed diffuse dilatation at the coarctation site (balloon:coarctation ratio of 4·5). At follow up all patients were normotensive (only one on treatment). Reinvestigation at 6–16 (mean 12) months in 12 patients demonstrated residual gradients of 0–24 (5) mm Hg; one of these patients underwent successful redilatation.

Balloon angioplasty is effective in relieving native coarctation. The frequency of aortic dilatation at the site of the coarctation is small and may be related to balloon size. Our early experience with balloon angioplasty for native coarctation is encouraging. Its adoption as first-line treatment will depend on long term results and further experience.

Elective day case cardiac catheterisation: Judkins' technique

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Judkins' catheterisation is frequently performed as an inpatient procedure for fear of haemorrhage or other complications. A policy of day case catheterisation has been gradually adopted in this centre. During the 33 month period of this survey of 972 left heart studies (96% including coronary arteriography), including cases of recently unstable angina, 719 (74%) were performed electively as day cases; 43% had left main or triple vessel disease. Patients attended the cardiac department one hour before the procedure, which was performed without premedication using a French 7 or 8 sheath; patients taking warfarin were instructed to omit the treatment the day before the study. After catheterisation patients were observed in the day room of the cardiology ward for a minimum of four hours. 675 (94%) had uncomplicated procedures. There were two deaths (0·3%). 44 (6%) patients had to be admitted: eight with prolonged chest pain, seven with significant puncture site haemorrhage, six with acute infarction, and three with major vascular complications. Puncture site haemorrhage occurred within four hours except in one case (6 hours); it did not occur in any patient after discharge. The incidence of haemorrhage was unrelated to sheath size but was slightly more common in patients who had been receiving warfarin (4% *vs* 1%).

Day case femoral cardiac catheterisation can be performed with an acceptably low complication rate. Compared with a policy of admission there is less disruption of the catheterisation service by lack of beds; also a considerable cost saving and greater convenience for patients.

Provision of pre-hospital coronary care by a taxi service

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A mobile coronary care unit staffed and driven by a doctor and nurse was started in Dundee in 1977. During 1985 Tayside Health Board considered discontinuing the service because the van had been repeatedly involved in road traffic accidents. Since 6 January 1986 the unit has been conveyed by a local taxi firm. Patients are transferred to hospital by conventional ambulance. We have reviewed our initial 10 month experience of this novel service. There have been 458 calls, mean cost £5·33 each, compared

with approximately £8·00 for the previous doctor driven service in 1984. The mean distance travelled was four miles. 51% of requests were from general practitioners, 22% from ambulance-men, 8% from GP receptionists, 4% from other hospitals, and 15% directly from patients. The median time between requests for service and arrival at the patient was 14 minutes compared with 11 minutes previously. Drug treatment was given in 66% of calls; 65% of patients required hospital admission, 38% to a coronary care unit. During the first year of this service advanced cardiopulmonary resuscitation was attempted out of hospital on 18 occasions; however, only three cardiac arrests occurred in the presence of the team; two of these patients survived and remained well. There have been no road accidents.

The taxi based mobile coronary care unit can provide a low cost safe method of transporting a mobile unit team into the community.

Type A behaviour pattern in patients with coronary artery disease

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Possession of the type A behaviour pattern (TABP) is considered to be a risk factor in patients with coronary artery disease and may exert its effect by excessive stimulation of the sympathoadrenal medullary system. Additionally, type A subjects may perceive life events as more stressful than type B subjects. The frequency of the TABP in 30 patients undergoing routine angiography was studied by the Gough adjective check list and Thurston Activity schedule (these reflect time, pressure, and speed at which activities are undertaken). Forty eight hour ambulatory ST Holter monitoring was performed with concomitant collection of urinary catecholamine (nonadrenaline and adrenaline) during a stressful period in the patients lives and at a later date. The effect of possession of the TABP on the occurrence of myocardial ischaemia during daily life on these two separate occasions was studied. Patients kept a comprehensive diary of episodes of chest pain and emotional upset. Scores consistent with previously documented interview assessment of type A1 behaviour pattern were obtained in 12 (Gough) and 14 (Thurston) patients. There was good correlation between the questionnaires. Type A patients experienced many more episodes of painful myocardial ischaemia during the stressful period of monitoring than at the later date ($p < 0·02$). They also had a higher excretion of adrenaline ($p < 0·03$) at this time and reported more episodes of emotional upset compared with the later time ($p < 0·02$). There was no effect on the frequency of asymptomatic ischaemia.

Apolipoprotein AI/CIII/AIV DNA sequence markers, hyperlipoproteinaemia, and coronary artery disease in Scotland

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It is well recognised that in coronary heart disease (CHD) a family history is an important risk factor. Occasionally it is due to a mendelian genetic disorder such as familial hypercholesterolaemia but in most instances it is attributed to a combination of multiple genetic and common environmental factors. With recombinant DNA technology it is now possible to assess and analyse the genetic contribution in several diseases and already several reports suggest that variants of the genes that code for apolipoproteins (apo) are implicated in CHD. Using standard techniques we have identified allelic variants at five polymorphic DNA endonuclease restriction sites in the apoAI/CIII/AIV gene cluster and have determined their frequencies (*a*) in two Scottish populations ($n = 224$ and 130) with different standardised CHD mortality ratios (81 and 138), (*b*) in survivors of myocardial infarction in two coronary care units ($n = 62$ and 52), and (*c*) in patients assessed by coronary angiography ($n = 100$). Our results so far show a significant association between the frequency of allelic variants at a PstI site flanking the 3′ end of the apoAI gene and hyperlipidaemia (total cholesterol 7·5 mmol/l; triglycerides 2·1 mmol/l; $p < 0·005$), and with CHD before the age of 55 years ($p < 0·01$).

The findings are consistent with linkage disequilibrium between the apoAI/CIII/AIV gene marker and a gene or genes which confer predisposition to CHD in the Scottish population.

Neuropeptide Y causes myocardial ischaemia in man by small vessel constriction

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Peptidergic nerves appear to have a physiological role in cardiovascular regulation. Neuropeptide Y (NPY) is found in abundance in epicardial human coronary arteries in relation to noradrenergic nerves and is a potent constrictor of coronary arteries in isolated rabbit heart. We studied six patients with chest pain but no evidence of variant angina. Provocation tests including exercise, hyperventilation, ergometrine, and histamine were negative. All patients had either normal coronary arteries or only minimal wall irregularities, although one had evidence of previous myocardial infarction. During ECG monitoring, coronary arteriography was performed before and after control intracoronary infusions of 1% albumin in physiological saline and was repeated after five-minute intracoronary infusions of 0·2, 0·6, and 1·0 pmol/kg/min of NPY into the left (five patients) or right (1 patient) coronary artery. The epicardial vessel diameter was measured by computerised arterial analysis (CAAS-Pie Data Medical). Anginal pain with ST segment elevation occurred in 3 patients (two patients at 0·2 and 1 patient at 1·0 pmol/kg min). This was associated with interruption of flow in the infused artery as indicated by lack of progression of contrast. There was a slow return to a normal flow pattern after 2 mg of intracoronary isosorbide dinitrate. In the early phase of recovery when ischaemia had not completely resolved, lack of progression of contrast was still evident in the distal segment of the artery but there was no evidence in the proximal segment of focal constriction of the type seen in variant angina.

NPY produces myocardial ischaemia without epicardial vessel changes and therefore is a constrictor of small coronary vessels.

The influence of short term or long term aspirin administration after experimental vein graft insertion

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We have used a rabbit model of autogenous vein grafting to investigate the effect on graft intimal thickness of 40 mg/kg aspirin given daily for three days or for 28 days after insertion of a jugular vein graft into the carotid artery. Three groups of eight rabbits were grafted: untreated controls, three day treated, and 28 day treated. All animals were killed at 28 days (though one animal in the 28 day treated group died during the experimental period). Grafts were removed, fixed at pressure, and cut into seven cylindrical lengths. Intimal thickness was measured under microscopy with an Apple graphics tablet from elastin-van Gieson stained sections. A mean of 299 measurements was made on each section and the average intimal thickness was calculated for each graft. The mean thickness (μm (SEM)) for each group was: control 65·41 (8·23); three day treated 76·03 (7·37); 28 day treated 147·13 (79·60). Nonparametic analysis of the data indicated that, while there was no significant difference between the three day and 28 day treated groups, it can be stated with 95% confidence that the presence of aspirin was consistent with a reduction of up to 19% or an increase of up to 108% in intimal thickening.

While aspirin has proven beneficial effects in reducing the incidence of early postoperative thrombosis, its potential, in a high dose, to produce greater intimal thickening, which may affect the long term survival of the graft, must also be considered.

Coronary anatomy and collateral vessels during evolving acute myocardial infarction

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We investigated the relation of symptoms and coronary arteriographic findings in 65 consecutive patients aged 27–78 years presenting with acute myocardial infarction. Coronary arteriography was performed before administration of thrombolytic treatment at a mean of 4·0 (range 1·1–7·9) hours after the onset of continuous chest pain.

Twenty three patients gave a history of chronic stable angina pectoris and 21 of recent onset unstable angina; 21 had no history of pain before infarction. The infarct related coronary artery was occluded in 58 patients and patent with a severe stenosis in seven; in 37 it was the only coronary obstruction present. In 45 patients collateral vessels were seen filling the infarct related coronary artery, but they were present with partial or complete filling of the infarct related artery in 20. There were no significant differences in the frequency of collateral filling of the infarct related artery between those patients with (seven of 23) or without (13 of 32) chronic angina. Multiple vessel obstruction, particularly three vessel disease, was more common in patients with chronic stable angina ($p < 0·05$).

Patients with acute myocardial infarction most commonly have single vessel disease. Collateral vessels rarely fill the infarct related artery during the early phase of infarct evolution and are no more frequent in patients with preceding chronic angina.

Pulmonary embolectomy: indications and results

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Between 1964 and 1986 139 patients with acute massive pulmonary embolus (AMPE) were admitted to this hospital. Before 1968 pulmonary embolectomy was the only specific treatment available and was performed on 30 patients, with a mortality of 33·3%. Although the introduction of thrombolytic treatment as an alternative treatment reduced the annual number of pulmonary embolectomies this operation was still required in 41 of 109 patients admitted with AMPE (37·6%) either because the patient was too sick to allow a trial of thrombolytic treatment (5 cases) or because there was a specific contraindication to thrombolysis (30 cases) or operation was considered to be the treatment of choice (4 cases). Two patients started on streptokinase but later required emergency embolectomy. Thus over the whole period 71 pulmonary embolectomies were performed: of these, 21 (29·6%) patients died. Sixteen of those who died had required preoperative external cardiac massage (ECM), indicating the severity of their haemodynamic impairment. Three of these 16 died on bypass, nine died within four weeks with severe neurological damage, three died in the first postoperative week of circulatory or multisystem failure, and one patient died at five weeks with carcinomatosis. Of the five patients who died without having had preoperative ECM one died on bypass, one died of pleural and one of gastrointestinal bleeding, one patient died within 24 hours of persistent hypotension, and one at six days with massive venous occlusion requiring thrombectomy. An additional patient died five months after operation with a further pulmonary embolus. Fifty (70·4%) of the 71 patients survived to leave hospital. One underwent a period of haemodialysis and then made a full recovery, three had moderate neurological deficits which were thought to have been present preoperatively, and one developed a hemiplegia perioperatively, probably caused by paradoxical embolus through a patent foramen ovale. The remaining survivors (n=45) recovered completely. Out of the total group of 71 patients undergoing pulmonary embolectomy 25 had required preoperative ECM. In the latter patients the mortality was 63·6% compared with 12·2% in patients who had not required ECM.

We conclude that pulmonary embolectomy is still an important and effective form of treatment for AMPE.

Sudden death in hypertension: a possible mechanism

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Sudden death is common in hypertensive patients with electrocardiographic left ventricular hypertrophy (ECG LVH) and is often attributed to myocardial infarction secondary to otherwise silent coronary artery disease. In other forms of cardiac hypertrophy, however, primary ventricular arrhythmias occur frequently and are predictive of subsequent sudden death. We investigated the occurrence of ventricular arrhythmias by 48 hour ambulatory ECG monitoring and their relation to coronary artery disease in hypertensive patients with and without ECG LVH and in a normotensive control group. Ventricular tachycardia (VT) ($\geq$3 ventricular impulses at a rate >120 per minute) occurred in 1/50 (2%) control subjects, 4/50 (8%) hypertensive patients without LVH, and 14/50 (28%) hypertensive patients with LVH ($p<0·01$ *vs* controls, $p<0·05$ *vs* hypertensives without LVH). Coronary arteriography in 16 of those with VT demonstrated normal coronary arteries in nine (56%) and single vessel disease in four (25%). Left ventricular endomyocardial biopsy specimens from those patients with VT were analysed by point counting and compared with biopsy specimens from patients with a similar degree of LVH but without VT on ambulatory monitoring. Volume fractions of interstitium and of fibrosis were 19(4)% and 13(3)% respectively for those with VT and 3(1)% ($p<0·001$) and 2(1)% ($p<0·001$) for those without.

Ventricular arrhythmias occur commonly in hypertensive patients with left ventricular hypertrophy; their occurrence cannot be attributed solely to coexistent coronary artery disease, but is related to myocardial complications of left ventricular hypertrophy, including myocardial fibrosis. They may contribute to the high frequency of sudden death in hypertensive patients.

Aortic valve replacement with and without myocardial revascularisation in patients with coronary artery disease

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To assess the role of myocardial revascularisation in patients with combined aortic valve disease (AVD) and coronary artery disease (CAD) early and late outcome were determined in 630 patients who underwent aortic valve replacement between 1974 and 1982. Three groups of patients were identified. Group I (506 patients) did not have significant CAD (>50% reduction of intraluminal diameter of a major coronary artery), group II (69 patients) had CAD and underwent coronary artery bypass grafting, and group III (55 patients) had CAD but did not undergo myocardial revascularisation. Patients in group II had more severe CAD than those in group III: 10 had left main stem stenosis, 17 had three vessel disease, 19 had two vessel disease, and 23 had single vessel disease, compared with 2 patients, 2 patients, 16 patients, and 35 patients respectively in group III. The overall early mortality (within 30 days of operation) was 6% for group I, 13% for group II, and 16% for group III, although operative mortality was lower in patients operated on more recently (3%, 8%, and 13% respectively). The three year survival of patients in group I (83%) was significantly higher ($p < 0.001$) than that of patients of group III (62%) but not than that of patients in group II (76%).

The findings suggest that the presence of CAD increases the risk of aortic valve replacement whether or not coronary artery grafting is performed. Myocardial revascularisation, however, appears to return patients with AVD and CAD to a survival curve similar to that of patients with isolated AVD.

Sudden death; occurrence in 300 cases of sarcoid heart disease in the United Kingdom

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This study investigates sudden death in the first 300 cases of sarcoid heart disease collected by the author over 15 years in the United Kingdom: 279 were white and 21 were black; 162 were male and 138 female; and the average age was 48 years. One hundred and thirty seven have died; 103 had necropsy examinations. Sudden death occurred in 77: (*a*) it was the method of presentation in 49 and (*b*) it occurred in 28 patients already diagnosed as having sarcoid heart disease. Of the 49 cases first diagnosed at necropsy, 29 were male and 20 were female, and 43 were white and 6 black. Most of the postmortem examinations were coroner's cases and they were often incomplete; however in all of them sarcoid granulomas were present in the myocardium and other organs. Massive heart involvement was frequent and the initial macroscopic label was of tumour. Occasionally the heart was unremarkable until examined microscopically. Twenty eight of the cases were from East Anglia and the rest were scattered throughout the UK, suggesting that local awareness of sarcoid heart disease was important. Twenty eight known cases of sarcoid heart disease died suddenly and 26 had postmortem examination. Nine of these examinations were considered to be substandard. Fourteen cases were known to the author in life and this interest led to the correct postmortem diagnosis finally being made. Some pathologists are reluctant to consider sudden death as being due to causes other than coronary artery disease. The commonest clinical signs of sarcoidosis were intrathoracic. In four out of 17 cases tested the Kveim test was negative. The type of cardiac presentation, treatment, and survival are considered.

Sarcoid heart disease is not rare; it is an occasional cause of sudden death and it may be overlooked even at necropsy.

The effect of aircraft vibration on the function of an activity-sensing pacemaker

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The new generation of rate-responsive pacemakers includes devices incorporating a vibration sensor which influences the pacing rate relative to the perceived level of user activity. This study examined the effects on the function of such a device of extraneous vibration such as that encountered in aircraft. A bipolar Activitrax pacemaker (Medtronic, Minneapolis, USA) was attached externally to the pectoral area of a normal subject and programmed to typical settings (basic rate 60 beats/min, maximum activity rate 150 beats/min, medium activity threshold, rate response 5). The pacing rate was monitored by a Medtronic 5309 pacing system analyser incorporating a digital display and recorded every 15 seconds during flights in six fixed wing aircraft, four helicopters, and a hovercraft. In the fixed wing aircraft the maximum observed pacing rates were 115 beats/min in a light twin, 92 beats/min in a twin turboprop, and 79 beats/min in a large passenger jet. Significant increases in rate were confined to brief periods such as taxying, takeoff, turbulence, and landing, with only modest rises during most of the flights. In the helicopters and hovercraft sustained rate rises were noted throughout the flights with

maxima of 100–131 beats/min. Re-setting the pacemaker activity threshold and rate response level achieved only partial reduction of the maximum rate.

Vibration associated with air travel may increase the pacing rate of activity-sensing rate-responsiveness pacemakers. In fixed wing aircraft such increases are only transient and minor but in helicopters and hovercraft they are considerable and sustained and may cause difficulty in some patients.

Comparison between maximal exercise treadmill testing and tournament squash in men over 45 years

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Treadmill stress testing is used routinely in the evaluation of cardiac symptoms experienced during competitive exercise. There are few studies comparing the physiological response to exercise in these two circumstances. We have compared the cardiovascular and metabolic responses to exercise in maximal treadmill exercise testing and competitive squash in 10 men (>45 years). Subjects were studied at rest before exercise, on completion of exercise, and then at five and 30 minutes after exercise. The following were measured on each occasion: heart rate rhythm; potassium; catecholamines (adrenaline, noradrenaline); lactate; glucose; free fatty acids; and haematocrit. A paired Student's t test was used for statistical analysis. Treadmill exercise was associated with a higher mean (SEM) maximal heart rate (178 (3) *vs* 165 (4); $p<0.05$), higher mean plasma lactate (7·4 (0·5) *vs* 5·2 (0·6 mmol/l; $p<0.0025$), and higher mean plasma potassium (4·1 (0·2) *vs* 3·6 (0·14) mmol/l; $p<0.05$) than squash at the end of exercise. Five minutes after squash mean serum free fatty acids were raised (0·72 (0·14) mEq/l) and mean plasma potassium was below the normal range (3·2 (0·8) mmol/l). Mean blood glucose and mean plasma catecholamines tended to be higher in squash. There was no difference in mean haematocrit between squash and treadmill exercise.

We conclude that treadmill exercise is a good reflection of the cardiovascular responses occurring during competitive squash. However, squash is associated with changes in potassium and free fatty acids which may be arrhythmogenic.

Cryoablation of the accessory pathway in Wolff-Parkinson-White syndrome: initial results and long term follow up

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Cryoablation of the accessory pathway (AP) has been used in the management of Wolff-Parkinson-White (WPW) syndrome where surgery is indicated. We examined the outcome in 17 patients who were then followed up for a mean of 5·7 (range 1–9) years. There were 10 women and seven men aged 21–58 years (mean 38·6). Indications for operation were failure of medical treatment for reentrant atrioventricular tachycardia in 11 patients and atrial fibrillation with rapid anterograde conduction across the AP in six. Thirteen patients had overt preexcitation but it was concealed in four. The AP was located on the left free wall in nine, posteroseptally in four, on the right free wall in two, and anteroseptally in one. In one patient two APs were located on the right—one anteriorly and the other posteriorly. There was one postoperative death from rupture of an unsuspected cerebral haemangioma and one patient suffered hemiparesis. In six patients there was recurrent arrhythmia, one after one day, one after two weeks, two after one month, and two after five months. In these patients the APs were located on the left free wall in four, posteroseptally in one, and anteroseptally in the other. Reoperation was successful in four of the six; two are still managed medically, one with an anti-tachycardia device. Of the 16 survivors, 14 remain arrhythmia free.

Successful cryoablation of the AP is possible in WPW syndrome and, if arrhythmia recurs, further surgery is usually successful. Initial operative failure does not seem to be related to the site of the AP but may reflect the epicardial approach used with this technique. If surgical cure is not achieved interference with the AP may be sufficient to render the arrhythmia more amenable to medical management.

Left ventricular repolarisation sequence in man

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The repolarisation sequence of intact human heart is

unknown. It is widely postulated that repolarisation proceeds in the opposite direction to activation, but hitherto it has not been possible to test this hypothesis. The epicardial repolarisation sequence was studied intraoperatively in eight patients with upright T waves undergoing coronary artery bypass grafting. Monophasic action potentials were recorded from left ventricular sites (13–25 sites/patient) with a hand held probe. The left ventricle was considered as four concentric rings between base and apex. Mapping was performed during fixed rate right atrial pacing on normothermic cardiopulmonary bypass. Action potential duration increased by 30–40 ms at all epicardial sites in the first minutes of bypass. Mapping was started on return to an approximate steady state. Activation occurred earliest at the apex and latest at the base (variance analysis, $p < 0{\cdot}001$; apex − base = 18 ms (5) SEM). The action potential duration was inversely related to activation time and was longest at the apex and shortest at the base (variance analysis, $p < 0{\cdot}025$; apex − base = 16 ms (9) SEM). However, there was no significant difference in repolarisation time between the four rings (apex − base = 2 ms (5) SEM).

These findings differ from the classically postulated repolarisation sequence. They show that epicardial repolarisation is independent of activation sequence and occurs simultaneously between base and apex. The observed inverse relation between activation time and action potential duration may represent a mechanism for preserving the homogeneity of repolarisation.

Treatment with ambulatory dobutamine infusion of severe chronic heart failure refractory to current treatment

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Severe chronic heart failure often requires repeated and prolonged admission to hospital. Thus in the absence of effective treatment to improve survival, an improvement in the quality of life is a genuine therapeutic aim. The use of dobutamine infusion in acute heart failure is well established. Therefore, we undertook an open study of continuous ambulatory dobutamine in four patients (49–60 years) with severe chronic heart failure (New York Heart Association Class III and IV) who had remained symptomatic (minimum three months), and required repeated admission to hospital despite diuretic, cardiac glycoside, and vasodilator treatment. All four had benefited from acute dobutamine and understood and participated in catheter pump care. Dobutamine (7 μg/minute) was administered via a Hickman central venous catheter by a portable infusion pump over a 48 hour period. Patients attended as outpatients for refilling of the pump at the end of each period. One patient died in hospital after eight weeks (acute pulmonary oedema) and three patients were followed up for 24–43 weeks. All four patients reported a reduction in dyspnoea, fatigue, and personal incapacity. The number of inpatient days was reduced from a mean of 64 days per patient per six month period to 0·75 days per patient per six month period. There was an improvement in clinical signs, New York Heart Association classification by at least one class, and in mean treadmill exercise duration from 3·6 to 9·75 minutes. Resting left ventricular ejection fraction assessed by radionuclide ventriculography showed no significant change. No increase in tachycardia was detected by serial Holter recording and there were no major infective or mechanical complications.

Preliminary results at six months indicate that supervised continuous ambulatory dobutamine reduces symptoms and hospital admissions, while increasing functional capacity in severe heart failure, without adverse effect on cardiac rhythm or major infection.

Left ventricular end systolic volume is the major determinant of survival after recovery from myocardial infarction

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Impairment of left ventricular function is the major predictor for mortality after acute myocardial infarction, but it is not known whether this is best described by ejection fraction or by end systolic or end diastolic volume. We measured volumes, ejection fractions, and severity of coronary arterial occlusions and stenoses in 605 male patients (< 60 years old) 1–2 months after a first (n = 443) or recurrent (n = 162) myocardial infarction, and we followed these patients for a mean of 78 months (range 15–165 months). There were 101 cardiac deaths. Multivariate analysis using log rank testing and the Cox proportional hazards model showed that end systolic volume ($\chi^2 = 82{\cdot}9$) had greater predictive value for survival than end diastolic volume ($\chi^2 = 59{\cdot}0$) or ejection fraction ($\chi^2 = 46{\cdot}6$), while stepwise analysis showed that once the relation

between survival and end systolic volume had been fitted there was no additional significant predictive information in either end diastolic volume or ejection fraction. Severity of coronary occlusions and stenoses showed additional prediction of only borderline significance ($p=0.04$ in one analysis). For a subset of patients ($n=200$) who had taken part in a randomised trial of coronary artery surgery after recovery from infarction, surgical "intention to treat" showed no predictive value.

We conclude that left ventricular end systolic volume is the primary predictor of survival after myocardial infarction. Treatment of infarction should be aimed at limitation of infarct size and prevention of ventricular dilatation.

Improved left ventricular function and survival after acute myocardial infarction with intravenous streptokinase: a double blind trial with and without concomitant intravenous propranolol

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Streptokinase 1.5×10^6 units or placebo was infused intravenously over 30 minutes within 4 hours (mean 3 hours) of the onset of infarction in 219 patients under 70 years of age. In 44% who did not have contraindications, intravenous propranolol followed by oral propranolol was also given, while heparin, low dose aspirin, and dipyridamole were given to all. Cineangiocardiograms were performed at three weeks after infarction in 98% of eligible patients; ejection fraction (EF) and end systolic volume (ESV) were calculated, and contractility was scored from biplane ventriculograms. Thirty day actuarial survival was improved by streptokinase (4/108 deaths among streptokinase patients *vs* 14/111 in placebo patients; $p=0.02$). Survivors from first infarction who had had streptokinase showed higher EF (59 (1)% (SEM) *vs* 53 (2)%; $p<0.005$), lower ESV (55 (3) ml *vs* 73 (5) ml; $p<0.005$), and better contractility score (2·9 (0·3) *vs* 4·8 (0·4); $p<0.01$) than those who had had placebo. Patency of the infarct related artery was 78% after streptokinase and 56% after placebo. Subgroups with anterior or inferior infarction and those who had or had not had propranolol showed similar significant benefit from streptokinase.

We conclude that intravenous streptokinase is an effective treatment and that the combination of streptokinase with intravenous propranolol is well tolerated.

Randomised trial of high dose intravenous streptokinase, of oral aspirin, and of intravenous heparin in acute myocardial infarction

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619 patients with suspected acute myocardial infarction (MI) were randomised to receive either a high dose short term intravenous infusion of streptokinase (1·5 MU over one hour) or placebo. In addition, using a "$2 \times 2 \times 2$ factorial" design, patients were also randomised to receive either oral aspirin (325 mg on alternate days for 28 days) or placebo, and separately randomised to receive either intravenous heparin (1000 IU/hour for 48 hours) or no heparin. Streptokinase (SK) was associated with a non-significant decrease in hospital mortality (7·7% in those allocated to SK *vs* 9·2% in those allocated to placebo) and an increase in non-fatal reinfarction (3·6% *vs* 2·9%). There were significantly more minor adverse events after SK (for example hypotension, allergies, bruises, or minor bleeds), but no excess of strokes or of anaphylactic shock. Aspirin was associated with fewer reinfarctions (2·9% in those allocated to aspirin *vs* 3·9% in those allocated to placebo; NS), deaths (6·1% *vs* 10·5%; $2P<0.04$) and strokes (0·3% *vs* 2·0%; $2P<0.1$). Heparin was associated with a decrease in reinfarction (1·9% in those allocated to heparin *vs* 4·9 in those allocated to no heparin; $2P<0.04$), though not in mortality (8·3% *vs* 8·2%; NS), and with a trend towards more strokes (1·6% *vs* 0·7%; NS) and more bruising and bleeding 14% *vs* 11%; NS). To assess reliably the effects of SK and aspirin on major endpoints several hundred hospitals are now collaborating in a large (about 20 000 patients planned) randomised trial (ISIS-2).

Cardiopulmonary response to dynamic exercise after combined heart-lung transplantation

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To determine the effect of combined heart-lung transplantation (HLT) and the resultant cardiac and pulmonary denervation on the cardiopulmonary response to dynamic exercise, we studied eight HLT recipients during symptom limited graded exercise

on a cycle ergometer and compared them with eight recipients of orthotopic cardiac transplant (CT) and eight normal subjects (N) matched for age and sex. Heart rate, ECG, blood pressure, ventilation, and oxygen saturation (ear oximeter) were monitored and expired gas analysis performed with a computerised mass spectrometer. All data are quoted as mean values (SD); p values refer to comparisons with normal subjects. The maximum oxygen uptakes (l/min) of the groups were: N 1·96 (0·44); HLT 1·33 (0·45), $p<0·05$; CT 1·5 (0·62) NS. Heart rate rose with oxygen uptake in all three groups and the slopes of the regression lines (beats/litre) were: N 57·7 (15·3), HLT 28·8(14·8), $p<0·01$; CT 24·9 (13·7), $p<0·01$. Ventilation rose with oxygen uptake and the slopes of the regression lines (below the anaerobic threshold) were: N 23·9 (5·5), HLT 30·8 (6·1), $p<0·05$; CT 25·8 (3·2), NS. Oxygen saturation did not fall significantly during exercise. Systolic blood pressures (mm Hg) at rest were N 119 (9), HLT 108 (17), CT 108 (14) and at maximum exercise N 176 (20), HLT 146 (19), $p<0·05$; CT 148 (22), $p<0·05$. The maximum oxygen uptake of both HLT and CT recipients was lower than that of normal controls.

Both transplant groups have an abnormal heart rate response to exercise; this is related to cardiac denervation. HLT recipients also show an increased ventilatory response at submaximal workloads, which may reflect altered ventilatory control after pulmonary denervation.

Arrival-time imaging of the thoracic aorta: a new way of demonstrating the site of aortic dissection

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Intravenous digital subtraction aortography (DSA) can accurately detect dissection of the thoracic aorta. Although the intimal flap can usually be distinguished from similar linear subtraction artefacts by its characteristic motion throughout the cardiac cycle, further analysis avoiding image subtraction is an obvious advantage. Since digital images are composed of a large number of pixels it is possible to compute time-density curves for each pixel throughout the period of the study. One static image depicting the time to 90% maximum density for each pixel is then constructed. Structures which do not change in density by more than 10% throughout the study, such as the background, are excluded, avoiding the need for image subtraction. Structures enhanced by contrast are depicted on a colour scale dependent on the arrival time of the contrast. Therefore, provided contrast arrives later in the false lumen, it is possible to demonstrate both the presence and, more importantly, the site of entry of the aortic dissection. This technique provides a static image of aortic dissection, which, used in conjunction with image subtraction, helps to overcome subtraction artefact. All patients with suspected aortic dissection now undergo DSA with additional arrival-time analysis.

Atrial flutter: the relation between ECG flutter waves and endocardial atrial activity and excitability

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Spontaneously occurring human atrial flutter is most commonly manifest by its surface ECG appearance of sawtooth flutter waves in the inferior leads. In 71 consecutive cases of spontaneously occurring atrial flutter, "common" flutter was present in 63 (89%). Four cases exhibited "uncommon" flutter (positive F waves in leads I and II), and in three cases no F waves were visible. Endocardial mapping was carried out in 53 cases. 220 sites were mapped (46 high anterior or lateral right atrium (HRA), 67 mid anterior or lateral (MRA), 70 low anterior or lateral (LRA), and 38 septal RA or coronary sinus). In 16 cases, 19 sites demonstrated fractionated or split electrograms (10 MRA, 9 LRA), consistent either with local block or slow activation. Split activation was more likely to occur in the mid or low anterolateral RA ($p<0·005$). Lateral RA wall activation was determined in 39 cases of common flutter (133 sites), and was upwards in seven, downwards in 27, and discordant (suggestive of local block) in five. Split potentials were present in 12 of these cases, unassociated with activation pattern. In uncommon flutter (11 sites, 4 cases), lateral RA was activated upwards in two, downwards in one, and discordantly in one. Three split electrograms were seen. When septal activation could be determined (eight cases), it was codirectional with lateral wall activation in four and opposite in four. Atrial activation was seen throughout the flutter cycle, but more RA sites (42%) were activated during the second half of the surface F wave than at any other part of the cycle. Atrial stimulation was carried out during atrial flutter at 54 sites. Fully compensatory (type II) pauses after atrial extrasystoles were seen at 17 sites

and reset (type I) was observed at 37 sites. Type I response was seen most often at LRA sites (12/14), and less often at MRA (11/17), HRA (7/11), and septal (7/12) sites ($p < 0.02$). No definitive evidence for a common site of the macro reentry circuit in human atrial flutter was found. However, split or fractionated electrograms were only seen in the mid and low anterolateral RA, and were particularly associated with type I sites in the LRA. Thus it appears that the reentry circuit in human atrial flutter is more likely to involve this portion of the right atrium. This finding suggests that it may be possible to direct ablative treatment to this area.

For how long after cessation does smoking continue to act as a risk factor for the development of coronary heart disease and does the in-hospital course of a first myocardial infarction differ in non-smokers, ex-smokers, and current smokers?

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We studied 978 consecutive patients admitted with a first myocardial infarction or episode of acute coronary insufficiency to examine differences between non-smokers, ex-smokers, and current smokers in risk factor profile, presentation, and in-hospital outcome. Non-smokers were older than either ex-smokers or current smokers (mean ages 65·6, 62·8, and 60·6 years respectively ($p < 0.01$). Hypertension was more common in non-smokers ($p < 0.01$) who also had a higher mean serum cholesterol concentration ($p < 0.05$). A stepwise logistic regression model selected age and hypertension as independently discriminating non-smokers from current smokers ($p < 0.01$). Applying this function to ex-smokers, patients who had stopped smoking less than 15 years before admission had a similar risk factor profile to current smokers, while those who had stopped for at least 15 years had a profile resembling non-smokers ($p < 0.05$). Non-smokers were more likely to have anterior infarctions than inferior ones ($p < 0.05$), but they had similar peak cardiac enzyme activities to ex-smokers and current smokers. Their in-hospital mortality (16·7%) was significantly higher ($p < 0.01$) than that in ex-smokers (7·2%) or current smokers (5·4%) They also had higher rates of left ventricular failure ($p < 0.01$) but not of arrhythmias or of conduction defects. When adjusted for age, site, and size of infarction and occurrence of complications, non-smokers still showed a significantly higher death rate ($p = 0.025$).

Ex-smokers may carry an increased risk of acute coronary heart disease for up to 15 years after stopping smoking. Non-smokers are older than ex-smokers or current smokers at the time of their first episode and their higher mortality may reflect more severe underlying coronary artery disease.

Plasma atrial natriuretic peptide: haemodynamic and hormonal inter-relations in cardiac impairment

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To explore the inter-relations of plasma atrial natriuretic peptide (ANP), renin, and aldosterone concentrations and haemodynamic function in cardiac impairment, we studied 63 unselected patients with cardiac lesions of a broad spectrum of type and severity undergoing diagnostic cardiac catheterisation. Plasma ANP was measured by radioimmunoassay after Sep-Pak extraction from plasma. Plasma active renin and aldosterone concentrations were also measured by radioimmunoassay. Inter-relations between plasma ANP, renin, and aldosterone concentrations were assessed both in normal subjects and in the patient group. Plasma concentrations of ANP correlated closely with New York Heart Association functional class ($r = 0.77$, $p < 0.001$), right atrial pressure ($r = 0.83$, $p < 0.001$), pulmonary arterial wedge pressure ($r = 0.65$, $p < 0.001$), and pulmonary arterial pressure ($r = 0.70$, $p < 0.001$). Among normal subjects plasma ANP concentrations were inversely related to concurrent concentrations of renin ($r = -0.52$, $n = 140$, $p < 0.001$) and aldosterone ($r = -0.38$, $n = 140$, $p < 0.001$). Plasma renin and aldosterone values correlated positively ($r = 0.55$, $n = 140$, $p < 0.001$). In contrast with the inverse relation between plasma ANP and both plasma renin and aldosterone seen in normal subjects, in the patients with cardiac impairment this relation was lost and replaced by positive associations between ANP and both renin ($r = 0.31$, $n = 60$, $p < 0.02$) and aldosterone ($r = 0.63$, $n = 57$, $p < 0.001$). However, the relation of renin to aldosterone ($r = 0.49$, $n = 58$, $p < 0.001$) was not disturbed by the very high plasma concentrations of ANP found in cardiac impairment.

ANP rises progressively with cardiac impairment. Normally plasma ANP and renin-aldosterone acti-

vity alter in a reciprocal fashion. Concurrent renin-aldosterone system activation and an increase in plasma ANP is a feature of severe cardiac disease and may reflect concurrent increases in intra-atrial pressures together with impaired renal perfusion.

Does angiotensin converting enzyme inhibition reduce myocardial ischaemia in patients with stable angina?

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Angiotensin converting enzyme inhibition reduces left ventricular diastolic pressure, aortic systolic pressure, and sympathetic drive. Such effects should reduce myocardial work and oxygen consumption and should be beneficial to patients with angina. To test this hypothesis, enalapril (10–20 mg/day) was evaluated in 12 normotensive patients (age 46–70 years mean 55) with angina and exercised induced ST segment depression. The study was randomised, double blind, placebo controlled and crossover with each treatment period lasting two weeks. Assessment was by angina diaries and maximum symptom limited treadmill exercise testing (modified Bruce protocol). All medication except glyceryl trinitrate was stopped seven days before the start of the study. The results for the group as a whole showed a reduction in mean (SD) systolic blood pressure at rest from 134(16) to 118(15) mm Hg ($p < 0.05$) and at peak exercise from 165(30) to 146(27) mm Hg ($p < 0.05$). Heart rate at rest and at peak exercise was unchanged. Frequency of angina attacks and consumption of glyceryl trinitrate were unaltered by enalapril. Total exercise duration on enalapril increased from 466(94) to 509(115) seconds (NS) and exercise duration to 1 mm ST depression from 345(77) to 387(114) seconds (NS). By contrast with these findings for the group, four patients had striking prolongation of total exercise time (mean 450 to 591 seconds) and exercise duration to 1 mm ST depression (mean 315 to 435 seconds).

Although enalapril was not effective in reducing myocardial ischaemia in the group as a whole, some patients were substantially improved.

Provocation of ventricular tachycardia by high energy catheter ablation of normal atrioventricular conduction

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Catheter ablation of normal atrioventricular (AV) conduction is now the treatment of choice for many refractory supraventricular arrhythmias. Although it is successful in about 90% of patients a few have subsequent ventricular tachycardia (VT) and sudden death. Because these might have been caused by the ablation procedure we prospectively searched for VT in 11 of our patients undergoing catheter ablation of normal AV conduction for refractory supraventricular arrhythmias. All patients had ambulatory ECG recording before and after ablation and were studied during programmed ventricular stimulation by a standard stimulation protocol immediately before and 2–4 days after the procedure.

No patient died during a follow up (15 to 26 months (mean 20·5)). None of the ambulatory ECGs showed VT before or after ablation. In eight patients VT was not inducible at either stimulation study. Non-sustained VT was inducible in one patient with equal facility before and after the ablation. VT occurred spontaneously in two patients within 24 hours of the ablation. Emergency cardioversion was required in one but VT was not inducible at the stimulation study performed three days after ablation and has not recurred. In the other patient with early spontaneous VT, the same VT was reproducibly initiated at subsequent stimulation studies and this patient required prophylactic treatment 25 months afterwards. One patient had asymptomatic non-sustained VT (4 beats) documented once during follow up eight months after ablation.

VT may be unpredictably provoked by high energy catheter ablation of normal AV conduction and usually presents soon after ablation; it should be sought and may require longer term treatment.

A longitudinal study of smooth muscle cell turnover within and platelet deposition on experimental vein grafts

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Intimal thickening is an important cause of the long term failure of vein grafts. We used a rabbit model to assess the time course of platelet deposition and smooth muscle cell turnover in grafted vessels. In separate operations, one or two weeks apart, according to group, each animal received two jugular vein grafts into the carotid artery. Groups of rabbits were killed three and five weeks and three and six months later and the grafts were harvested. Twenty four

hours before death autologous platelets labelled with indium-III tropolonate were injected and 45 minutes before death each rabbit received a pulse dose of tritiated [3H] thymidine. Measurement of platelet deposition on one graft was derived from indium-III counts of the vessel and a blood standard. Platelets $\times 10^6$ g graft tissue (mean (range)) in each group were: three weeks 244 (87–472), n=5; five weeks 189 (111–317), n=7; three months 101 (52–218), n=6; six months 111 (65–154), n=6. One three week old graft and one six month old graft were blocked; two grafts were contaminated with postmortem clot and these were not measured. Intima was stripped mechanically from the other graft and the [3H] thymidine uptake per μg DNA was compared with the mean measure obtained from the abdominal and thoracic aortas of the same animal. The ratio graft: aorta (mean (range)) in each group was: three weeks 16·9 (5·2–27·6), n=6; five weeks 11·6 (6·2–17·0), n=4; three months 2·2 (0·6–4·8), n=7; six months 2·0 (0·9–3·6), n=6. In three grafts, one each from the five week, three week, and six month groups, no intima or turnover could be detected. The activity of smooth muscle cell turnover rose and fell in parallel with platelet deposition. At six months while platelet deposition appears to have reached a steady state, the rate of smooth muscle cell turnover in the grafted tissue was still double that observed in the aorta. Any treatment that might be directed against smooth muscle cell turnover in order to reduce graft intimal thickening therefore needs to continue for at least six months after grafting.

Morphology of coronary arterial lesions and early thrombotic occlusion after percutaneous transluminal coronary angioplasty

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Early thrombotic occlusion occurred in eight (24%) of 33 consecutive patients who intitially had successful percutaneous coronary angioplasty (PTCA) for unstable angina. Five of these had successful repeat PTCA and three had emergency coronary bypass grafting. This unexpectedly high event rate led us to analyse the coronary artery lesion morphology to determine whether it was possible to predict those at risk from early thrombotic occlusion. The coronary artery lesions were retrospectively analysed by two angiographers who did not know the outcome of the PTCA. The coronary lesions were classified into three groups according to their complexity and irrespective of the severity of the stenosis. Eight patients had symmetrical smooth lesions (type 1), nine patients had asymmetrical smooth lesions (type 2), and sixteen patients had more complex asymmetrical, irregular, or "punched out" lesions (type 3). Clinical characteristics were similar in the three groups. One of the eight patients with type 1, one of the nine with type 2, and six of the 16 with type 3 lesions had early thrombotic occlusion.

Although PTCA is an effective treatment for unstable angina patients with unstable angina and angiographic complex lesions are at risk from early thrombotic occlusions after PTCA.

Possible pitfalls when mean aortic valve gradient alone is used to grade aortic stenosis

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Echocardiographic and Doppler estimation of mean aortic valve gradient (MAVG) compares well with invasive measurements. Although MAVG gives an approximate assessment of the severity of aortic stenosis misclassification may occur with abnormal stroke volumes. This is a particular problem when echocardiographic and Doppler determination of stroke volume is unreliable, for example in mixed aortic valve disease or left ventricular dilatation. Gorlin's formula for aortic valve area (GAVA) incorporates stroke volume and is accepted as a good measure of the severity of aortic stenosis. Therefore, we compared MAVG with GAVA to see how frequently MAVG alone would misclassify aortic stenosis. 636 patients with aortic stenosis (with or without regurgitation) who had undergone full cardiac catheterisation were reviewed. Patients with mitral valve disease were excluded. MAVG and systolic ejection period were averaged over at least five beats. Cardiac output was determined by dye dilution unless there was significant aortic regurgitation, in which case angiographic data on ventricular volume were used. GAVA was calculated and then compared with MAVG. We looked for a value of MAVG which could be used as a sensitive and specific predictor of critical aortic stenosis (GAVA $< 0{\cdot}75$ cm^2). With MAVG > 30 mm Hg there were few false negatives (16/165 (9%)) but many false positives (139/471 (29%)), giving a sensitivity of 95% and a specificity of 48%. MAVG > 60 mm Hg gave far fewer false

positives (26/210 (12%)) but more false negatives (164/426 (38·5%)) with a sensitivity of 53% and specificity of 91%. All patients with MAVG > 110 mm Hg had critical aortic stenosis and no patient with MAVG < 20 mm Hg had critical aortic stenosis. However, these limits included only 11% of patients. These results demonstrate the importance of measuring stroke volume in all patients when echocardiographic and Doppler are used to evaluate aortic stenosis.

Technetium–99m pyrophosphate myocardial SPECT is a promising technique for sizing acute infarction. The technique is not influenced by the site of infarction and may have advantages when interventions that alter myocardial enzyme release kinetics are used. Further studies in the setting of intervention with tissue plasminogen activator are planned.

Infarct sizing: comparison of technetium–99m pyrophosphate tomography and creatine kinase–MB release

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Measurement of the extent of myocardial necrosis provides a major predictor of morbidity and mortality after infarction and is important for the assessment of interventions to limit infarct size. Release of myocardial creatine kinase (CK–MB) is used as a reference technique but release kinetics may be affected by interventions such as thrombolysis. Imaging acute infarction with technetium–99m pyrophosphate offers qualitative information but the determination of infarct volume by single photon emission tomography (SPECT) should provide quantitation of the extent of necrosis. We have studied 20 patients admitted to a coronary care unit within six hours of the onset of acute infarction. The extent of myocardial necrosis was determined by measurement of CK–MB release over 96 hours after the onset of symptoms. Planar and SPECT pyrophosphate myocardial imaging was undertaken between 24 and 72 hours after infarction. In 17 patients with uncomplicated acute infarction a good correlation was demonstrated between cumulative CK–MB release and the volume of myocardial pyrophosphate uptake measured by SPECT ($r = 0{\cdot}92$). Similar correlations were obtained for eight patients with anterior infarction ($r = 0{\cdot}90$) and nine patients with inferior infarction ($r = 0{\cdot}94$). SPECT provides a more accurate measurement of the extent of myocardial necrosis than that obtained from planar imaging and offers advantages in terms of infarct localisation. Three patients were given intravenous streptokinase within four hours of the onset of major symptoms and substantial alterations of CK–MB release kinetics were demonstrated. In this limited study the relation between cumulative CK–MB release and myocardial pyrophosphate uptake appeared to be maintained.

Significance of left ventricular gradient in hypertrophic cardiomyopathy

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The significance of left ventricular (LV) gradients in hypertrophic cardiomyopathy (HCM) is controversial. To assess the proportion of stroke volume left in the left ventricle at the onset of systolic anterior motion (SAM) of the mitral valve we performed concurrent technetium–99m radionuclide cineangiography and colour flow mapping with continuous and pulsed wave Doppler echocardiography in 41 consecutive patients with HCM. Twelve had complete SAM with septal contact, 14 had incomplete SAM without septal contact, and 15 did not have SAM; none had moderate/severe mitral regurgitation. The peak velocity of blood flow was significantly greater in complete SAM (3·8 (1) m/s *vs* incomplete 1·8 (0·3) and *vs* no SAM 1·4 (0·3) m/s). Radionuclide peak ejection rates (3·9 (0·7), 3·9 (0·8) and 3·9 (1·0) EDV per s and ejection fraction (78 (9), 76 (8), and 75 (7) per cent) were similar. The time from the R wave to the onset of SAM septal contact was 232 (37) ms. The proportion of stroke volume ejected by SAM septal contact in patients with complete SAM was 48–89%, median 72 (14), and after 230 ms in patients with incomplete SAM and no SAM it was 70 (10) and 73 (14) per cent respectively. In patients with complete SAM the magnitude of the LV gradient was inversely related to the proportion of stroke volume ejected ($r = 0{\cdot}71$, $p < 0{\cdot}01$).

These findings suggest that HCM with or without a LV gradient is associated with rapid, early, and near complete systolic emptying, but in a minority, particularly those with early onset of SAM and large gradients, there is considerable residual stroke volume after SAM septal contact that is consistent with "obstruction".

Serodiagnosis of *Streptococcus faecalis* endocarditis by immunoblotting of surface protein antigens

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Endocarditis caused by *Streptococcus faecalis* is becoming more common, particularly in elderly men, and the condition remains difficult to treat. Surface protein antigens, expressed by serum-grown bacterial cells, have been used as the basis for a serodiagnostic test for *S faecalis* endocarditis. Three major species specific surface protein antigens, molecular weights 73, 40, 37 kd, can be demonstrated after separation of bacterial components by sodium dodecyl sulphate polyacrylamide gel electrophoresis followed by electrophoretic transfer on to a nitrocellulose membrane. The antigens are incubated with serum from patients with endocarditis and the antibodies that react with antigens on the membrane are visualised with a protein A-horseradish peroxidase conjugate. Antibody to at least one of the species specific antigens was detected in the serum of all six patients with *S faecalis* endocarditis who were tested. Serum antibodies to antigens prepared from other species of streptococci were not detected in any of these patients. Serum from 129 patients with various infections did not react with any of three species specific antigens. These included serum from patients with endocarditis caused by microorganisms (32), a septicaemias (34), *S faecalis* infections at other sites (50), and patients undergoing continuous ambulatory peritoneal dialysis (13). False positive reactions were seen in serum from three patients, however; one patient had had an episode of *S faecalis* endocarditis six months before collection of serum for the test; one had prolonged *S faecalis* colonisation of a nephrostomy tube, and one had endocarditis caused by a nutritionally variant streptococcus.

Prognostic criteria in post-infarction ventricular septal defect

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Mortality from post-infarct ventricular septal defect can be reduced by operation, selection for which would be helped by knowledge of factors affecting postoperative prognosis. We reviewed our eight year experience of 38 surgically treated patients, and compared the preoperative characteristics in those who died postoperatively (14) with those who survived (24, all still alive). Sex, age, infarct size (peak creatine kinase), left ventricular end diastolic pressure, pulmonary:systemic flow ratio (3·1 (0·8) *vs* 3·0 (0·7), mean (SE)), and blood urea were similar in the two groups. Non-survivors had significantly more inferior myocardial infarction (57 *vs* 21%), cardiogenic shock (64 *vs* 29%), right ventricular coronary perfusion graded (on the basis of assessment, blind of outcome, of angiographic anatomy, site of occlusion or stenosis, and overt collateral supply) as bad (8/11 *vs* 1/19), and higher right ventricular end diastolic pressure (15·0 (1·0) *vs* 11·2 (1·1) mm Hg). Of the nine with inferior infarcts who had angiography, four graded as having bad right ventricular perfusion all died; only one of the other five died (from incidental haemorrhage). Of the 21 patients with anterior infarcts by angiography, four of the five graded as having poor right ventricular perfusion died; only two of those graded as having good right ventricular perfusion died (one from perioperative inferior infarct and one with grossly impaired left ventricular function).

Prospective evaluation of factors predictive of outcome singly or in combination may help to identify those patients most likely to benefit from operation. The data show the importance of right ventricular function. The angiographic findings raise the possibility that prognosis might be improved by revascularisation or mechanical support or both where right ventricular perfusion is compromised.

Recombinant tissue plasminogen activator in the early phase of acute myocardial infarction

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Thirty six consecutive patients (30 men, 6 women; mean age 58 years (42–71)) were randomised at 20–240 minutes (mean 140) from onset of pain to receive 20, 50, or 100 mg of recombinant tissue plasminogen activator (rt-PA) by intravenous infusion over 90 minutes. At 90 minutes coronary angiography was performed. Fifteen patiens had grade 3, 6 had grade 2, nine had grade 1, and six had grade 0 reperfusion of the infarct related artery (0 vessel occluded, 3 distal flow as rapid as in a non-infarct related artery). Grade 2 or 3 reperfusion occurred in four of the 12 on 20 mg, seven of the 12 on 50 mg,

and ten of the 12 on 100 mg. All nine patients given rt–PA up to 80 minutes after the onset of pain had ⩾ grade 2 reperfusion. Thirty four of the 36 patients received a further 50 mg rt–PA iv over 1–5 hours. Heparin was then started in 33 patients and continued for 1–10 days. During hospital admission three patients developed clinical signs of reocclusion proven in two at repeat angiography. Gated blood pool scans in 27 patients at an average of 43 hours (16–104) after the onset of pain and regional third ejection fractions (RTEFs) calculated for septal, inferior, and posterior segments from the left anterior oblique 45° views showed that the infarct related RTEF for 13 patients with grade 2 or 3 reperfusion was 50·3 (4·98%) (mean (SEM)) whereas for patients with grade 0 or 1 reperfusion it was 32·3 (5·1%). Peak creatine kinase MB ranged from 31 to 1009 and peak total creatine kinase from 302–24 060 U/l (normal CK–MB up to 25 and CK 140 U/l). During rt–PA there was a slight reduction in serum fibrinogen. No significant bleeding occurred during rt–PA, and during heparin one patient required one unit of blood. During hospital admission, four patients died from cardiogenic shock.

rt–PA (100 mg) is a most effective thrombolytic agent. When it was administered within 80 minutes of onset of chest pain rt–PA allowed reperfusion regardless of dose. Reocclusion, bleeding complications, and reduction in serum fibrinogen were minimal.

Haemodynamic response to ibopamine: evidence for differential activity on the venous and arterial sides

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Ibopamine is an orally active dopamine derivative which has been used as a positive inotrope in patients with congestive cardiac failure (CCF). The haemodynamic effects of an oral dose of 200 mg ibopamine were measured invasively in eight patients who had significant left ventricular dysfunction (pulmonary capillary wedge pressure ⩾15 mm Hg). A thermodilution balloon catheter was placed in the pulmonary artery to record right atrial pressure (RAP), pulmonary arterial pressure (PAP), wedge pressure (PCWP), and cardiac output (CO). The aortic and left ventricular pressures were measured and the contractility indices dP/dt and dP/dt p′ derived. Baseline values were recorded and measurements were made every 15 minutes for an hour after dosing. A rise in cardiac output from 5·9 (1·6) l/min to 7·5 (1·1) l/min ($p < 0·05$) was seen at 45 minutes with a concomitant rise in heart rate from 73 (9) beats/minute to 93 (24) beats/minute ($p < 0·01$). The systemic vascular resistance showed an early fall from 1435 (357) pre-dose to 1227 (351) dyne s^{-1} cm^{-5} at 15 minutes which was sustained at one hour. However, an early rise in right sided venous pressures was seen at 15 minutes, the RAP rising from 4 (2) mm Hg to 7 (2) mm Hg ($p < 0·05$) and returning to baseline values at one hour. Similar profiles were seen with mean PAP (23 (8) mm Hg to 34 (10) mm Hg at 15 minutes ($p < 0·001$) and mean PCWP (14·6 (7) mm Hg to 25 (13) mm Hg at 15 minutes ($p < 0·01$)). There was a smaller rise in left ventricular end diastolic pressure at 15 minutes from 27 (5·5) mm Hg to 33 (6·0) mm Hg. The derived indices of contractility, dP/dt and dP/dt p′, showed a moderate rise at 45 minutes (1356 (254) pre-dose to 1854 (589) and 22·1 (4·8) pre-dose to 24·6 (3·4) respectively (NS)). These results suggest a differential effect on venous and arterial sides which may be explained by selective pulmonary venoconstriction or differential stimulation of α and β receptors.

The need for coronary arteriography in patients with aortic valve disease

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It has been suggested that because of improvements in non-invasive assessment of patients with aortic valve disease (AVD) preoperative cardiac catheterisation may be unnecessary, particularly if there is no history of chest pain. It is known that some patients with AVD have significant coronary artery disease (CAD) despite the absence of chest pain. How large is this group and does it have any distinguishing features? Over a 12 year period 921 adults underwent cardiac catheterisation to assess the severity of AVD. Right and left heart catheterisation and coronary arteriography were performed in all cases. Patients with mitral valve disease were excluded. 481 patients had angina or atypical chest pain and 170 (35·3%) of these had CAD. 440 patients gave no history of chest pain; 73 (16·6%) of these patients had CAD. Comparison of the subgroup without chest pain with the overall group showed that patients without pain were older ($p < 0·00001$) and had a higher incidence of syncope ($p < 0·0007$). Differences in other risk factors including sex, family history of CAD, other vascular disease, smoking, diabetes, and hyperlipidaemia, how-

ever, were not significant. In the group without pain the youngest man was aged 40 and the youngest woman 56. In the total group there were 242 patients aged <50 of whom 12 men and two women had CAD. No patient aged <40 (n=100) had CAD although two had important coronary artery anomalies. In the group of patients without chest pain aged >40 (n=378) 19·3% had CAD.

These findings suggest that even when there is no history of chest pain there is an appreciable frequency of CAD in patients with AVD aged >40. At present coronary arteriography is the best method of identifying this group.

Myocardial capillary basement membrane thickness in young diabetics

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Previously we have reported an impaired ejection fraction response to exercise in young diabetics. To investigate the contribution of small vessel disease to this abnormality of ventricular function, we compared endomyocardial right ventricular biopsy specimens from seven symptom free type 1 (insulin dependent) diabetic patients aged 30–50 years with biopsy specimens from seven non-diabetic age and sex matched controls examined by light and electron microscopy. The diabetic patients had normal coronary arteriograms but all showed a reduced ejection fraction on exercise. Myocardial capillary basal laminar thickness was measured by electron microscopy. No significant difference in basal laminar thickness was demonstrated between diabetic (mean 79·3 nm) and control patients (mean 73·0 nm). Light microscopy showed significant but non-specific interstitial fibrosis in five diabetic patients and arteriolar thickening in two diabetic patients.

Thus impaired ventricular function in young asymptomatic type 1 diabetics is associated with significant histological abnormalities. These, however, are not related to coronary artery disease or to capillary basement membrane thickening which has been described as a characteristic feature of diabetic capillaries in several other organs including kidney, skin, and skeletal muscle.

Rapid termination of supraventricular tachycardia in children by adenosine

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The purine nucleoside adenosine is an effective antiarrhythmic agent in man, impairing atrioventricular nodal conduction and thereby terminating reentrant supraventricular tachycardia (SVT). Three neonates and one older child (aged 10 years) presented with seven episodes of SVT refractory to other agents. The neonates were all in cardiac failure because of prolonged SVT associated with the presence of an accessory pathway. The older child underwent electrophysiological study for investigation of recurrent atrioventricular nodal tachycardia. In each case adenosine was given intravenously in increments of 0·05 mg/kg, up to a maximum of 0·25 mg/kg. Termination of tachycardia was achieved in each case within 20 seconds of injection (range 9–20). No change in blood pressure was observed in any instance. In two patients inspection of the ECG immediately after termination of the tachycardia allowed a definitive diagnosis of the mechanism. The presence of atrial echo beats in one child and anterograde conduction down an accessory pathway in another (the appearance of delta wave on the ECG) provided evidence of a potential reentry circuit.

Adenosine is a safe and effective drug for the diagnosis and short term termination of SVT in severely ill children. The very short half life in human blood (10–20 seconds) allows rapid and accurate dosage titration and ensures that any possible side effects are transient.

Haemodynamic changes during early human pregnancy: an M mode and Doppler echocardiographic study

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Many of the haemodynamic changes that occur during pregnancy are thought to occur during the first trimester but the size and timing of these changes have not been adequately documented. We have used cross sectional, M mode, and Doppler echocardiography to study the changes in cardiac haemodynamic function during the first trimester of pregnancy. Twelve women were recruited before conception. Investigations were performed on day 21 of two consecutive menstrual cycles. After conception, investigations were repeated 5, 8, and 12 weeks after the last menstrual period. Mean (SEM) cardiac output has increased from 4·85 (0·11) l/min to 5·42 (0·11) l/min by five weeks ($p<0·001$). This increase was primarily due to an increase in heart rate from 74 (1) to 79 (1) beats/min ($p<0·01$). By 12 weeks cardiac output had increased to 6·58 (0·11) l/min ($p<0·001$) due to a further increase in heart

rate to 83 (1) beats/min ($p < 0.001$) and an increase in stroke volume from 65·1 (1·0) to 79·9 (1·9) ml ($p < 0.001$). End diastolic left ventricular dimension and left atrial dimension had increased by eight weeks ($p < 0.001$). Myocardial contractility, as measured by M mode echocardiography, had increased by five weeks ($p < 0.001$).

Cardiovascular adaptation occurs earlier in pregnancy than previously reported. Echocardiography is ideally suited to the measurement of haemodynamic changes during pregnancy.

Atrioventricular relations during tachycardia: are they useful for diagnosis?

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Analysis of the temporal relation of atrial and ventricular depolarisation may allow recognition of tachycardia by implanted dual chamber devices especially if atrioventricular or ventriculoatrial block is present. Difficulty arises when there is a 1:1 atrioventricular relation in tachycardia. We therefore examined the temporal relations of atrial (A) and ventricular (V) electrograms obtained at clinical electrophysiology studies during sinus rhythm and 58 episodes of junctional reentry tachycardia (JRT) from 58 patients aged from 6 to 72 (mean 38·2) years; 26 had ventricular preexcitation. There were 28 episodes of atrioventricular nodal reentry tachycardias, 30 of atrioventricular reentry tachycardias with 18 left (four concealed), 10 septal, and two right sided accessory pathways. If sinus rhythm was not preexcited, the AV interval was corrected to JRT rate by Lepeshkin's formula (SRc) but the AV interval was assumed to remain constant at all rates with preexcited sinus rhythm. The ratio of AV:VA intervals present during JRT was then compared with those during sinus rhythm of JRT rate.

AV:VA ratios (mean and range) for SRc *vs* JRT were respectively: 0·51 (0·33–0·81) *vs* 16·5 (2·7–70) for atrioventricular nodal reentry tachycardias; 1·47 (0·24–4·25) *vs* 1·94 (0·5–4·25) for atrioventricular reentry tachycardias with septal accessory pathways; 1·16 (0·7–1·61) *vs* 1·78 (0·96–3·15) for atrioventricular reentry tachycardias with overt left accessory pathways; 0·77 (0·64–0·91) *vs* 1·69 (1·17–2·13) for atrioventricular reentry tachycardias with concealed left accessory pathways; and 0·77 (0·29 and 0·48) *vs* 1·62 (1·14 and 2·09) for atrioventricular reentry tachycardias with right accessory pathways. AV:VA ratio analysis reliably allowed distinction between atrioventricular nodal reentry tachycardia and sinus rhythm with wide differences always present. Individual and overall similarities of AV:VA ratios in preexcited sinus rhythm and atrioventricular reentry tachycardias make distinction of these unreliable by this method.

Rate responsive pacing: is there already a dual sensor system?

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Respiratory dependent pacemakers sense regional variations in chest wall impedance and increase the heart rate in response to exercise induced hyperventilation. We have observed disproportionate increases in heart rate during certain forms of exercise, suggesting that the sensor also responds to non-respiratory movement. To test this hypothesis, ECG telemetry was used to record the heart rate response to a range of activities in five patients with complete heart block and respiratory dependent pacemakers (Biotec RDP3). Heart rate rose from 72(2) beats/min, mean (SEM)) at rest to 115(6) when patients were walking normally but fell to 88(6) during continued walking with restricted arm movement. Voluntary hyperventilation while standing still caused heart rate to rise to a peak of 96(7) beats/min. Heart rate increased to 119(11) beats/min when the arm ipsilateral to the pacemaker was swinging regularly but only reached 88(4) when the contralateral arm was swung.

The sensitivity of the respiratory sensor to arm movement should be taken into account when programming the RDP3 pacemaker and may profoundly affect the heart rate response to different forms of daily exercise. Appropriate signal processing may enable a single sensor to respond to both respiration and activity. These could then provide the inputs for a dual sensor rate responsive algorithm.

A transseptal aortic balloon valvotomy

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Aortic valvotomy by balloon catheter offers the hope of at least temporary relief of severe stenosis in patients unable to undergo valve replacement. Two major technical problems have been encountered when performing aortic valvotomy by the retrograde approach. The balloon position becomes unstable during inflation and removal of the deflated balloon through the arteriotomy causes local damage and consequent difficulty with haemostasis. An alternative technique has been devised that uses the femoral venous route to pass a guide wire across the atrial septum to the left ventricle and through the aortic valve. The dilatation catheter, single balloon type, is then passed over the wire and across the aortic valve. Traction applied to the catheter easily prevents systolic propulsion of the balloon into the aorta during inflations to 4 bar. Initial clinical experience showed that this technique overcame both problems and was successful in reducing the transvalvar gradient by >50% with no aortic regurgitation. There were no difficulties in crossing the septum with the balloon catheter and no subsequent shunt at atrial level.

We conclude that the transeptal venous route may be preferable for aortic valvotomy. Further favourable experience may widen the indications for the procedure and justify a randomised trial to compare its results with those of surgery in elderly patients.

A comparison of the effects of substance P and calcitonin gene related peptide on resistance vessels and veins in man

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The localisation of neuropeptides in the cardiovascular system has suggested a physiological role for peptidergic nerves in cardiovascular regulation. We compared the effects of substance P and calcitonin gene related peptide (CGRP) on resistance vessels, by measuring forearm blood flow by venous occlusion plethysmography during brachial artery peptide infusion, and also on the tone of superficial veins at rest after preconstriction by noradrenaline or after constriction stimulated by the single deep breath reflex. Incremental doses of CGRP caused a dose dependent increase in forearm blood flow (from 3·28 (0·56) to 9·08 (2·12) ml/min/100 ml forearm volume at 10 pmol/min ($n = 6$, $p < 0·01$, analysis of variance). Substance P caused a dose dependent increase in forearm blood flow from 2·18 (0·39) to 6·14 (1·72) ml/min/100 ml at 1·5 pmol/min. During a 30 min infusion of substance P the initially increased flow decreased, suggesting tachyphylaxis; increased flow caused by CGRP was maintained. The biological effective half life of CGRP was 18 minutes while that of substance P was approximately 15 seconds. Venous infusion of substance P did not affect resting venous tone but it relaxed those veins constricted by noradrenaline and also inhibited the venoconstrictor reflex. The lowest effective dose of substance P was 100 fmol/min but there was rapid development of tachyphylaxis and a dose response curve could not be constructed. CGRP had no effect on the veins.

CGRP and substance P have different patterns of onset and offset of actions and tachyphylaxis, suggesting different modes of action on blood vessels, though both may be involved in the control of vascular smooth muscle tone.

Good Hearted Glasgow: The Greater Glasgow Health Board multiple risk factor intervention programme for cardiovascular disease

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Scotland now has the highest standardised mortality rate (SMR) for coronary heart disease (CHD) in the world. The Glasgow SMR is 10% higher than that in the rest of Scotland. 45% of all deaths in Glasgow are caused by CHD and stroke. The daily average bed use in Glasgow for the management of all aspects of CHD and stroke is 1294 of which 418 are in the acute sector. The estimated hospital inpatient cost to the Health Board is over £30 million per annum excluding medical costs. The Glasgow MONICA Project has confirmed that over 75% of sudden deaths due to CHD occur before the victim can be admitted to hospital. In May 1985 the health board committed itself to a 10 year multiple risk factor intervention programme at an estimated overall cost of £8 million with the object of reducing the incidence and effects of CHD in Glasgow by 10% in 10 years. The strategy adopted is to provide a major community education programme about alterable risk factors and improved life style:

(a) By further developing the Glasgow 2000 Campaign to make Glasgow a smoking free city by the year 2000.
(b) By issuing dietary guidelines based on the Committee on the Medical Aspects of Food Policy Report, including animal fat reduction by 25% with substitution of unrefined carbo-

hydrate and fibre and promotion of increased physical activity.

(c) By providing, through general practice, screening of all Glaswegians aged between 20 and 50 (an estimated population of 400 600) for assessment of general health state, blood pressure, and blood cholesterol. Subjects with abnormal findings will be absorbed into the primary care and hospital system as appropriate. Following guidelines laid down in the Manual of Recommended Practice, individualised advice will be available to all persons screened.

(d) By developing community networks and special intervention for special groups.

The project and a pilot campaign covering 20% of the Glasgow population will be reported. Full coverage will be available by 1988.

Effects of lower body positive pressure on plasma atrial natriuretic peptide and sodium excretion in cardiac transplant patients

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Animal studies have suggested that the renal response to increased atrial pressure (a stimulus to atrial natriuretic peptide (ANP) release) is impaired after cardiac denervation. Recipients of cardiac transplants provide a model of cardiac denervation in man. Lower body positive pressure (LBPP) increases atrial dimension and circulating immunoreactive ANP (Ir-ANP) activity in healthy volunteers. We have studied the effect of LBPP on Ir-ANP, water, and sodium excretion in five cardiac transplant recipients (mean (SE) age 48 (4) years; mean (SE) time post-transplant 33 (10) months) and compared the data with those from 10 healthy volunteers (mean age 25 (1) years). All subjects followed the same protocol on each of two days; on one day LBPP was applied by inflation of a Medical Anti-Shock Trouser (MAST) suit to 40 mm Hg for one hour. Basal Ir-ANP was 58·7 (12·6) pmol/l in transplant patients and 7·0 (1·0) pmol/l in volunteers ($p < 0·01$). After 20 minutes of LBPP, Ir-ANP was unchanged in transplant patients but had risen to 11·1 (1·4) pmol/l in volunteers ($p > 0·05$ compared with basal). After 60 minutes of LBPP, Ir-ANP was increased in transplant patients (94·0 (15·1) pmol/l; $p < 0·05$) and remained increased in volunteers (11·6 (0·9) pmol/l). Thirty minutes after removal of LBPP, Ir-ANP had returned to basal values in both groups. Sodium excretion and urine flow in transplant patients were unaffected by LBPP but sodium excretion increased after LBPP (85·3 (9·2) to 117·2 (14·1) μmol/min; $p > 0·05$) and urine flow increased during LBPP (2·6 (0·5) to 6·7 (0·8) ml/min; $p < 0·05$) in volunteers.

These data confirm the increase in basal ANP in cardiac transplant recipients and suggest a blunting of the ANP response to LBPP. Furthermore, cardiac denervation in man appears to attenuate the natriuretic and diuretic response to LBPP.

Is ventricular fibrillation complicating acute myocardial infarction a reperfusion arrhythmia?

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Spontaneous reperfusion in acute myocardial infarction (AMI) is relatively common. For this reason it has been suggested that ventricular fibrillation (VF) complicating AMI may be a reperfusion arrhythmia. This suggestion was investigated by comparing profiles of creatine kinase (CK) release in VF patients with profiles in patients in whom the reperfusional state was determined angiographically. Nine patients with AMI who developed primary VF within four hours of the onset of symptoms were studied. Blood samples were drawn four hourly for CK estimation. The enzyme curves were compared with curves from 27 patients with AMI who received intravenous thrombolytic treatment within four hours of the onset of symptoms and underwent angiography within three hours of treatment. In patients with VF the mean (SD) time to peak CK was 24·3 (3·1) hours (range 19–29 hours). The time to peak activity of the CK-MB isoenzyme was similar (23·0 (2·7) hours), showing that cardioversion had not significantly altered the time course of enzyme release. In patients in whom the reperfusion state was angiographically defined the mean time to peak CK was 10·8 (2·9) hours (17 patients, range 6–15 hours) in those with a patent infarct related vessel, compared with 24·0 (5·1) hours (10 patients, range 16–31 hours) in those with persistent occlusion. The enzyme release profile in patients with VF therefore corresponded closely with that in patients with angiographically proven persistent occlusion.

Our results suggest that reperfusion is not a common cause of VF in AMI. As a corollary, the majority of patients with VF complicating AMI have not reperfused and may benefit from thrombolytic treatment.

Coronary atheroma evolution: patterns, correlates, and clinical implications

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We reviewed 77 patients who had had coronary angiography on two occasions. The mean interval between the studies was 21·6 months. Between the two studies there was a deterioration of symptoms in 55 patients (71%) and infarction in 15 (19%); while in 18 (23%) the symptoms were unchanged. Of the 274 lesions studied, progression was seen in 97 (35%), no progression in 144 (53%), and regression in 33 (12%). We identified 69 new lesions on the second study. No particular artery was susceptible to progression but lesions progressed more often in the mid segments than the proximal segments of vessels ($p < 0·05$). New lesions occurred uniformly throughout all segments of all vessels. A change in the mode of treatment was suggested in 43% of patients after the second study and 45% of those who were originally suitable for operation were either unsuitable or required a different graft pattern. In both these groups the interval between studies was longer than in the groups in which treatment was unchanged ($p < 0·05$). No significant correlation was seen between overall progression, defined as an increase in the Gensini score, and either interval or the risk factors smoking, hypertension, hyperlipidaemia, or family history of ischaemic heart disease.

Coronary atheroma evolves erratically and unpredictably without correlation with known risk factors. Clinical decisions need to be changed more often as the time from the initial study increases.

The value of exercise testing and thallium scintigraphy in identifying patients with myocardial ischaemia after thrombolytic treatment

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Patients who have undergone successful thrombolysis often have a severe residual stenosis of the infarct related artery. This may lead to ischaemia in the region of the infarct or further infarction if the vessel reoccludes. In order to identify these patients who may then require angioplasty or coronary artery surgery, we performed exercise testing and thallium scintigraphy in 58 subjects at a mean interval of 11·6 weeks after acute myocardial infarction which had been treated successfully by thrombolytic treatment. Coronary artery patency was confirmed angiographically and all patients had a severe residual stenosis (> 75%) of the infarct related vessel. Eleven (19%) patients had a positive exercise test. Twelve (20%) patients were found to have a "reversible" thallium perfusion deficit in the regions supplied by the infarct related vessel (six patients had both a positive exercise test and a reversible thallium perfusion deficit). Forty (69%) patients had a "fixed" perfusion deficit and the remaining six patients had no perfusion deficit (all inferior infarcts). There was no significant difference in the interval from the onset of symptoms of myocardial infarction to the administration of thrombolytic treatment between the reversible and fixed perfusion deficit groups (3·2 and 2·9 hours respectively). At the time of study six of the 12 patients with reversible thallium deficits had developed exertional angina and 12 of the 40 patients with fixed deficits had become symptomatic (correlation, NS); one patient in each group had confirmed reinfarction.

We conclude that exercise testing and thallium scintigraphy are of limited value in detecting residual myocardial ischaemia after thrombolytic treatment even in those patients with continuing symptoms.

Sectional Fourier analysis of gated cardiac tomograms

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A technique is described for the production and analysis of ECG gated tomograms of the left ventricle. Tomographic slices were produced with a multidetector emission tomographic scanner, after in vivo erythrocyte labelling with technetium-99m. ECG gated data were recorded in list mode and later used to construct a series of tomograms, which can be displayed in any projection as an endless loop movie for visual inspection. Further analysis involved the derivation of a series of short axis sections at right angles to the long axis of the left ventricle. Each was split into four segments and the amplitude, phase, and ejection fraction of each segment were calculated. For each segment and for each variable, a "normal" range was constructed from the data from 11 subjects with normal wall motion at cardiac catheterisation. The values of 13 subjects with abnormal wall motion at catheterisation were tested against this normal distribution. Abnormal values only were displayed on a bulls-eye representation of the ven-

tricle. There was complete separation of normal and abnormal subjects. Furthermore, the sectional analysis provided an accurate reflection of the site and severity of the wall motion abnormality.

Sex differences in the left ventricular response to exercise in patients with syndrome X

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Syndrome X, chest pain with evidence of ischaemia in patients with normal coronary arteries, remains a therapeutic and diagnostic challenge. Early reports suggested a female preponderance, but new evidence shows an equal sex incidence. To access whether there are any differences between the sexes in the response of the left ventricle to exercise we used first pass radionuclide angiography to study 32 patients (18 men, 14 women) with syndrome X. There were no significant differences between men and women in age, symptoms, or electrocardiographic abnormalities. Resting heart rate was significantly higher in women than men ($p < 0.05$), but exercise heart rate was not significantly different. Exercise duration in men was 6·6 (2·1) min and in women it was 4·1 (1·5) min ($p < 0.01$). Resting left ventricular ejection fraction was not significantly different (63 (10%) in men and 63 (9%) women), but on exercise there was a significant fall in women to 54 (10%) ($p < 0.01$ *vs* rest), but not in men (to 60 (15%), p not significant *vs* rest). Exercise left ventricular function was abnormal in seven men and 10 women and regional function was abnormal in four men and eight women. Only exercise duration had any significant linear correlation with left ventricular exercise response, but this was weak ($r = 0.41$, $p < 0.05$).

Abnormalities in exercise left ventricular function in a group of patients with syndrome X occur mainly in women and may reflect a difference in aetiology between the sexes. These differences suggest that the sex of the patient is important in the interpretation of non-invasive information in suspected coronary artery disease.

Failure of coronary anatomy to predict accurately left ventricular dysfunction on exercise

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To investigate the variable relation between the extent of coronary disease and the resultant left ventricular dysfunction on exercise, the results of first pass radionuclide angiography were reviewed in 135 patients with angiographic coronary disease and preserved left ventricular function. Fifty-one patients had one vessel disease, 37 had two vessel disease, and 47 had three vessel disease. Resting left ventricular ejection fraction was greater in one vessel disease (59 (8%) mean (SD)) than in two vessel disease (55 (9%), $p < 0.05$), or in three vessel disease (54 (9%), $p < 0.01$). Exercise left ventricular ejection fraction fell significantly in each group ($p < 0.001$) but the fall was less in one vessel disease (to 50 (9)%) than in three vessel disease (41 (12)%) ($p < 0.001$). There were no significant differences in exercise haemodynamic function, exercise duration, or change in left ventricular ejection fraction with exercise. Two additional indices that related change in left ventricular function to exercise duration and to heart rate slope were derived, but although there were some significant differences between the groups, the extent of overlap precluded accurate prediction. Multiple stepwise regression analysis showed exercise left ventricular ejection fraction to be the best and only independent predictor of coronary anatomy ($r = 0.35$, $p < 0.001$), but with a high degree of scatter.

There appears to be some relation between the extent of coronary obstruction and left ventricular dysfunction on exercise; however, a simple anatomical classification fails to predict accurately the extent of functional impairment. This suggests that studies of the effect of interventions in coronary disease should include a functional as well as an anatomical classification.

Floppy mitral and aortic valves: a common combination

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Over a 28 month period a classically floppy mitral valve was diagnosed in 117 new patients referred for echocardiography. Of these patients 50 had a normal aortic valve, 48 had echocardiographic features of a floppy aortic valve, and we identified intermediate features in 19. Doppler ultrasound (85 patients) showed no significant systolic gradients across any of the aortic valves: the frequency of aortic regurgitation in the 42 floppy, 32 normal, and 11 intermediate valves studied was 64·3%, 9·4%, and 0, respectively.

In patients with floppy mitral valves additional

myxomatous degeneration of the aortic valve is common and such valves are often mildly regurgitant.

Signal averaged ECG and Doppler aortic blood flow after myocardial infarction

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Transcutaneous continuous wave Doppler aortic blood flow and the signal averaged ECG were studied in 74 consecutive patients admitted after acute myocardial infarction (MI). Patients also had radionuclide or angiographic assessment of left ventricular function and 24 hour ECG. Patients with bundle branch block (5), atrial fibrillation (1), and complete heart block (1) were excluded from the signal averaged ECG analysis. A positive result (one or more of: filtered QRS > 120 ms, terminal QRS duration below 40 μV for > 40 ms, and RMS voltage during last 40 ms of < 25 μV) was present in 15/67 patients (22%). An abnormal signal averaged ECG was found more often in those with non-Q MI (8/23(35%) non-Q *vs* 7/39(18%) Q, $p < 0.05$). Peak enzyme activities were higher in those with an abnormal signal averaged ECG (aspartate transaminase 242(35) *vs* 174(13) IU/l and 2-hydroxybutyrate dehydrogenase 738(47) *vs* 556(36) IU/l, $p < 0.0001$). Doppler studies showed a significant correlation between maximum acceleration and the angiographic and radionuclide ejection fractions ($p < 0.001$, $r = 0.75$). The maximum acceleration was lower in patients with an abnormal signal averaged ECG (11(3) *vs* 16(4) $ms^{-1} s^{-1}$, $p < 0.001$). There was no correlation between the results of ambulatory ECG monitoring and the signal averaged ECG. During follow up, seven (10%) patients died; in one death was thought to be caused by pulmonary oedema and rapid atrial fibrillation and in another to reinfarction. Of the remaining five patients, who suffered deaths from arrhythmia, the signal averaged ECG was abnormal in four; 11 of the 67 survivors had an abnormal signal averaged ECG.

We conclude that: (*a*) transcutaneous Doppler is a useful indicator of left ventricular function and MI; (*b*) an abnormal signal averaged ECG is more common in patients with impaired left ventricular function after MI; (*c*) both techniques merit further study in this context because they are non-invasive, inexpensive, and easily performed, and they provide useful information which may enhance risk stratification in patients after myocardial infarction.

A quart into a pint pot: computer modelling to improve cardiac surgical throughput

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We have written a computer program to model the activity of a cardiac surgical unit. The number of operating sessions and beds, whether in intensive therapy unit (ITU) or wards, are defined constraints, and the user specifies a representative spectrum of patients to provide a data base from which the computer by simulation, admits, operates, and discharges cases. Throughput and bed occupancy figures are provided for each area of practice. Our unit has 16 beds, four days operating, and unpredictable use of 2–4 ITU beds. Day case work-up and early district hospital transfer were included in the model. The computer simulation achieved 310 cases with 92% bed occupancy and relative under usage of theatre (72%) and ITU (62%). In 1985 over 400 bypass cases, and additional closed operations, were performed, with ostensibly, the same constraints. In practice flexible management strategies were used to achieve the 90 extra cases. By using the model we explored the impact of admission of predictable postoperative problem cases late in the week and other selective admission policies, transfer of long stay cases (> 4 days) to general ITU beds, and the recognition of extra operating, arbitrarily timed on Saturday. When these were incorporated in the model, 410 cases were achieved.

Dipyridamole versus exercise thallium imaging: comparable or not?

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Although exercise is the conventional method of obtaining myocardial stress during thallium imaging, intravenous dipyridamole (DP) now provides an alternative and extends stress imaging to patients who exercise poorly. The two techniques are mechanistically different, however. A study was performed to compare the quality of the stress images obtained by both techniques to define better the role of DP stress imaging.

Exercise was performed according to a limited Bruce protocol test and DP was given by an intravenous infusion (0·5 mg/kg over 4 minutes). Thallium-201 (75 MBq) was injected 60 s before the

completion of treadmill exercise and in the DP group after gentle walking on the spot exercise. Images were obtained in the left anterior oblique (LAO), left lateral, and anterior views with a gamma camera and associated computer system. An image quality index was derived from the ratio of the maximum myocardial uptake segment to uptake in a fixed lung segment. The LAO view was used. Normalised thallium counts for both myocardium and lung were also compared.

The images of 30 consecutive DP and 30 exercise studies in the same time period were studied. The mean exercise duration in the exercise group was 7·33 minutes (range 3–12·66). The mean myocardial uptake of thallium was significantly higher in the DP group (140(45) *vs* 101(29) counts/s/unit area; $p < 0·001$); however, the lung-background uptake was also higher (69(22) *vs* 47(10) counts/s/unit area; $p < 0·001$). As a consequence the indices of image quality were not significantly different (2·06(0·29) *vs* 2·14(0·36); $p > 0·1$).

DP and exercise stress give similar quality thallium images. These technical data support the more widespread use of the DP stress technique.

Improved functional capacity in patients with hypertrophic cardiomyopathy treated by dual chamber pacing

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Work already presented to the British Cardiac Society has documented improvement of haemodynamic function and symptoms in patients with hypertrophic cardiomyopathy treated with dual chamber pacing (DDD). The present cooperative study was designed to compare functional capacity, as assessed by sequential exercise testing, in paced and in sinus rhythm. Fourteen patients have been entered in this study. Eleven patients have been followed for at least one year and the results of eighteen pairs of exercise tests on these patients are presented. In seven patients in whom repeated exercise tests were carried out, the order of testing was reversed on the second occasion. In all cases exercise was symptom limited and terminated by dyspnoea or fatigue or both. Mean (SD) treadmill endurance time was increased from 7·23(3·37) minutes to 9·74(3·86) ($p = 0·0012$). All eleven patients improved by at least one functional class as assessed by Canadian Heart Association criteria. Before pacing nine were in class 3 or 4 and 2 were in class 2. One year after pacing seven were in class 2 and 4 in class 1.

These observations demonstrate a significant and sustained improvement in exercise tolerance, and support the previously reported haemodynamic and subjective improvement in patients with severely symptomatic hypertrophic cardiomyopathy treated with dual chamber pacing.

Inappropriate use of intravenous verapamil in patients with ventricular tachycardia

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Intravenous verapamil is an effective treatment for supraventricular tachycardia (SVT) but it has also been used for broad complex tachycardia, where the diagnosis is uncertain. The response to intravenous verapamil in 29 episodes of ventricular tachycardia (VT), incorrectly diagnosed as SVT, was analysed. There were 17 patients (13 men, 4 women) aged 35–85 years (mean 59 years). Underlying heart disease was present in 15 patients: coronary artery disease in 12 and cardiomyopathy in three. The mean tachycardia rate was 190(25) beats/min (range 150–240 beats/min). The mean QRS duration was 0·15(0·01) s. Electrocardiographic features consistent with VT were present in all patients, and the diagnosis was confirmed by electrophysiological study in 12. Verapamil was administered in dosages ranging from 2·5 mg to 30 mg (mean 11·2(6·1) mg). In 23 episodes (79%) the tachycardia was not terminated, and in 13 of these severe hypotension occurred. Cardiac arrest occurred in two patients (caused by asystole in one and ventricular fibrillation in the other). The mean systolic blood pressure of those in whom the tachycardia persisted was 100(22) mm Hg before and 83(17) mm Hg after verapamil ($p < 0·01$). In 16 episodes DC cardioversion was required to terminate the tachycardia. No serious haemodynamic effects were observed in 12 episodes. In two patients without underlying heart disease, sinus rhythm was restored in four episodes. At least one serious adverse effect occurred in 10 patients (59%).

Verapamil is ineffective and potentially hazardous in VT and its use is inappropriate for the treatment of broad complex tachycardia unless a supraventricular origin has been clearly established.

Wolff-Parkinson-White syndrome: rapid ventricular response to atrial fibrillation in previously asymptomatic individuals

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Atrial fibrillation (AF) is a potentially lethal complication of the Wolff-Parkinson-White (WPW) syndrome; reentrant atrioventricular tachycardia (RAVT) is considered to be an important mechanism in precipitating it. Furthermore, when AF complicates WPW syndrome there is usually a history of prior symptomatic RAVT. We analysed the clinical presentation, electrocardiograms, and electrophysiological data of 108 consecutive symptomatic patients with the WPW syndrome. In nine (8%) AF was documented as the arrhythmia at the time of their first episode of palpitation. One presented with atrial flutter with 1:1 conduction. There were four women and six men. Though the mean age was 40 years, a bimodal age distribution was noted; four were 24 years or younger and six were 47 years or older. All complained of palpitation: three were presyncopal and three had syncope. Three had angina at presentation and one was hypertensive. Seven had type A WPW and three had type B. The range of mean ventricular rates observed clinically during AF was 125–259/min, whereas at electrophysiological study it was 173–310/min. The range of anterograde effective refractory periods of the accessory bundle was 230–290 ms.

AF may occur in the absence of antecedent RAVT, and dangerously high ventricular rates may be the first symptomatic manifestation of the WPW syndrome. The absence of a history or RAVT does not exclude the possibility of lethal arrhythmias. However, the role of prospective electrophysiology testing in symptom free patients, in whom the risk of sudden death appears to be minimal, remains to be established.

Intracavitary flow patterns in hypertrophic cardiomyopathy: a pulsed and continuous wave Doppler study with colour flow mapping

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Previous work has shown a direct relation between the extent of systolic anterior motion of the mitral valve (SAM) and measured intracavitary gradients in hypertrophic cardiomyopathy (HCM). We have now studied intracavitary left ventricular (LV) systolic flow patterns and velocity profiles in 47 consecutive patients with HCM using combined colour flow mapping with continuous and pulsed wave Doppler. Sixteen patients had complete SAM of the mitral valve, 14 had incomplete SAM, and 17 patients had no SAM. These were compared with the flow velocity patterns obtained from 10 normal individuals. All patients were in sinus rhythm. In normal individuals and patients without SAM colour flow mapping revealed laminar flow throughout systole with peak velocities of 0·65 (0·12) and 1·1 (0·2) m/s respectively ($p < 0·05$). In patients with incomplete and complete SAM there was laminar flow until the onset of SAM (106 (22) ms) when aliasing with colour reversal was seen at papillary muscle level; peak velocity at this level was 1·4 (0·5) m/s. After the onset of SAM septal contact (232 (37) all patients with complete SAM had a green-mosaic pattern of turbulent flow in association with an increase of peak velocity to 3·8 (1·1) ms.

Since the peak velocities were increased but similar in all patients before the onset of SAM, SAM cannot have been initiated by a Venturi effect as has been suggested, but such an effect could have been responsible for its later maintenance.